

LECTURE NOTES ON
Dermatology

BETHEL SOLOMONS

M.A. M.D. F.R.C.P.I.

*Consultant Dermatologist to Chelmsford and
Essex General Hospital, Herts and
Essex General Hospital and
Hertford County Hospital*

SECOND PRINTING

WITH 110 ILLUSTRATIONS
(32 IN COLOUR)

BLACKWELL
SCIENTIFIC PUBLICATIONS
OXFORD

This new title in the *Lecture Notes Series* sets out to provide the student, house physician and general practitioner with a clear survey of the important clinical features of all common skin diseases, and with concise notes on their treatment. It is especially designed for those whose experience has been slight, and whose acquaintance with the fundamental aspects of differential diagnosis of skin disorders is hazy. The methods of treatment described are restricted to those known to have definite value. The author stresses throughout that the skin is a multi-cellular and multi-organ structure: it is in this context that its importance, often as a reflector of systemic disease, is always indicated and emphasized where appropriate.

A notable feature of the book is the series of more than one hundred photographs, thirty-two of which are in full colour. These are an essential adjunct to any text on dermatology, and have been selected by the author with particular care to illustrate his clinical descriptions.

From the Reviews

'This new book is a notable addition to the Lecture Notes Series and it is wonderful value for money. . . . The book is attractively bound and clearly printed and, with its modest price, a good bargain for students and general practitioners.'—*The Practitioner*

'These lecture notes give a clear-cut picture of the more common skin diseases seen in general practice. The photographs are excellent and the text straightforward and very easy to read. . . . This is a most excellent book and can be thoroughly recommended.'—*Journal of the College of General Practitioners*.

616.5

Solomons

1

✓ RENFREW COUNTY LIBRARY

HEADQUARTERS: Marchfield Avenue, Paisley.

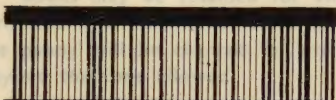
RESERVE

CARE OF BOOKS. Readers are asked to take great care of Library books in their possession.

2 079214 21

TIME ALLOWED FOR READING

weeks, and are due for return stamped below. A fine of one pence for every three days the book is kept beyond the limit.




RENEWALS. A loan may be extended if the book is not required by another reader.

REQUEST

13. 11. 76

REQUEST



Digitized by the Internet Archive
in 2022 with funding from
Kahle/Austin Foundation

LECTURE NOTES ON DERMATOLOGY

LECTURE NOTES ON
Dermatology

BETHEL SOLOMONS
M.A. M.D. F.R.C.P.I.

*Consultant Dermatologist to Chelmsford and
Essex General Hospital, Herts and
Essex General Hospital and
Hertford County Hospital*

REVISED REPRINT



BLACKWELL
SCIENTIFIC PUBLICATIONS
OXFORD

© BLACKWELL SCIENTIFIC PUBLICATIONS 1965

This book is copyright. It may not be reproduced in whole or in part without permission. Application with regard to copyright should be addressed to the publisher.

FIRST PUBLISHED IN 1965

REPRINTED 1966

616.5
SOLOMONS
RESERVE

COPY 1

207921421

A. 3-11-67

Printed in Great Britain by
A. R. MOWBRAY & CO. LIMITED
and bound by
THE KEMP HALL BINDERY, OXFORD

Contents

	PAGE
Preface	vii
1 Anatomy, Physiology and Pathology	1
2 History, Examination and Diagnosis	11
3 Treatment	23
4 Eczema and Dermatitis	39
5 Erythemato-Squamous Eruptions	58
6 Erythematous Eruptions	79
7 Diseases due to Physical or Chemical Substances	91
8 Psychological Factors in Skin Diseases	96
9 Bacterial Diseases	104
10 Fungal Diseases	119
11 Diseases due to Viruses	131
12 Bullous Diseases	148
13 Diseases due to Parasites	155

	PAGE
14 Disturbances of Pigmentation	162
15 Diseases due to Metabolic or Hormonal Disorders	166
16 Diseases due to Vascular Disorders	170
17 Systemic Diseases of Unknown Cause	176
18 Diseases of the Appendages	187
19 Tumours	210
20 Congenital and Hereditary Diseases	231
Bibliography	240
Examination Questions	241
Approximate Metric and Imperial Equivalents	242
Index	243

Preface

This book is intended to be an introduction to dermatology for students and general practitioners. It deals chiefly with common diseases, and to a lesser extent with some conditions which are often a sign of systematized syndromes. Pathology has been described only where it is considered it may be helpful in diagnosis, and only those treatments which have been shown to be useful are mentioned. Differential diagnoses are comprehensively covered within the scope of this book. For those interested in exploring the wider avenues of dermatology, and in learning about the less common skin diseases, a short list of references is included in the appendix.

The importance of the skin cannot be underestimated when it is considered that life is a continuous process of the interaction between the body and its environment, and that the skin surface is the medium through which most external stimuli are received. There are of course the stimuli which provoke the senses of the eyes, ears and nose, and the reactions of the mind to various stimuli, but the skin is the first receiving station for most phenomena which are a normal part of our existence.

The skin must be thought of as an organ in the same way as we think of the heart, brain or stomach, and not merely as a protective covering, sometimes requiring attention for what may seem to be minor disorders. The health of the skin is just as important for our organic vitality, as it is for any other organ in the body, whether it be, for example, acne or eczema; and conversely, our general vitality also affects the skin in dealing with the great number of external influences it encounters throughout life.

I am grateful to many people for their help with this book. Particularly, to Dr P.M. Deville and Dr Napier Thorne for their comments, and to Miss Margaret Laurance, for correcting grammatical errors.

In cases where my photographs did not adequately demonstrate certain clinical features, I have been fortunate enough to have liberal assistance from several colleagues; and I am therefore indebted to the following for their generous loan of photographs: Dr Martin Beare, Dr Geoffrey Hodgson, Dr Arthur Rook, Dr Brian Russell, and Dr Eric Waddington; also to the Institute of Dermatology, London University, Mr R.J.Lunnon A.I.B.P., F.R.P.S. of the Institute, and Mrs Mary Hutson of Chelmsford Hospital Photographic Unit.

The price at which this illustrated book is published would not have been possible but for the financial assistance of certain drug firms, and it is in this respect that I wish to thank the following for their great generosity: Genatosan Ltd, Imperial Chemical Industries Ltd, Miles Laboratories Ltd, Smith and Nephew Pharmaceuticals Ltd, E. R. Squibb and Sons Ltd, and Upjohn Ltd.

I am also indebted to Mrs Ann Hemsley for undertaking the tedious job of typing the manuscript.

Finally, I wish to acknowledge the patience and good humour of Mr Per Saugman of Blackwell Scientific Publications during the preparation of the book.

BETHEL SOLOMONS

11 Wimpole Street,
London W 1



CHAPTER 1

Anatomy, Physiology and Pathology

ANATOMY AND PHYSIOLOGY

Apart from its apparent function as an elastic envelope to walk about in, the skin has several other important qualities. It is waterproof and airtight, although some substances may be absorbed by it.

It acts as a form of thermostat, the heat of the body being regulated by the blood vessels and by sweating.

It protects underlying organs from physical, chemical and other injuries.

It acts as a relay station between external influences and internal organs by means of its tremendously complicated network of nerve terminals.

It acts as an organ of expression, betraying the innermost feelings of anxiety by sweating, fear by pallor, and anger by redness.

It is an important store for water, and contains 18–20 per cent of the total water content of the body.

Histology

The skin is divided into three principal layers:

1. the epidermis;
2. the dermis;
3. the subcutaneous tissue.

Epidermis

The *epidermis* is subdivided into four layers: (a) the basal layer, (b) the malpighian layer, (c) the granular layer, (d) the horny layer

(Fig. 1). (An extra layer, the so-called lucid layer, lies above the granular layer, and is found only on the palms and soles.) The cells in the basal layer gradually evolve into the cells of the horny layer, as they make their way to the horny surface, changing only in shape and size, as they pass through the malpighian and granular layer.

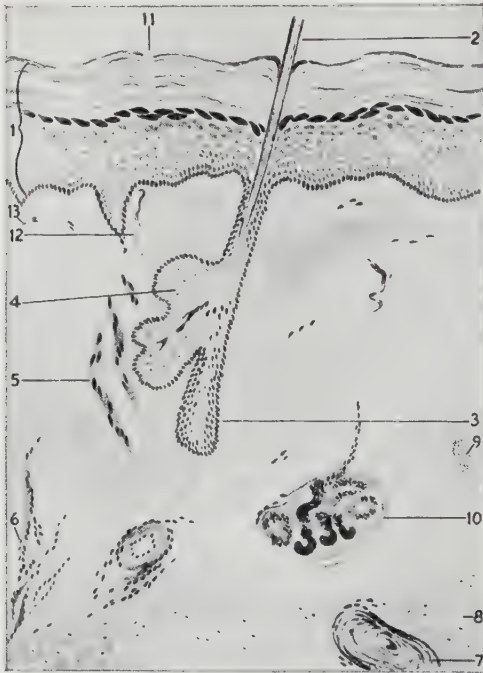


FIG. 1. Diagram of constituents of the skin.

1. Epidermis. 2. Hair. 3. Hair follicle. 4. Sebaceous gland. 5. Muscle fibres of *M. arrector pili*. 6. Blood vessel. 7. Pacinian body. 8. Fat lobules. 9. Cutaneous nerve. 10. Sweat glands. 11. Sweat duct opening. 12. Terminal nerve fibrils. 13. Collagen bundles.

The *basal layer* lies deepest in the epidermis, and next to the dermis. It consists of two types of cell:

- (i) basal cells,
- (ii) melanocytes.

Basal cells are columnar in shape, their long axis being at right angles to the dermis beneath them. They contain a dark staining oval or elongated nucleus which lies in deeply basophilic cytoplasm. They are joined to each other by intercellular bridges. Melanocytes appear as clear cells, with a small darkly-staining nucleus in clear cytoplasm; they lie at haphazard intervals between the basal cells.

The *malpighian layer* is also called the prickle cell layer, because the cells appear to be held together by prickles; they are actually intercellular bridges, which provide greater stability to the polygonal prickle cells.

The *granular layer* is composed of one to four rows of diamond-shaped cells, filled with deeply basophilic granules.

The *horny layer* normally contains no nuclei. Only by means of the electron microscope can intercellular spaces be seen. Its hardness is due to keratin (see p. 8).

The epidermis also possesses the following appendages:

- 1. the sweat, or eccrine, glands;
- 2. the apocrine glands;
- 3. the sebaceous glands;
- 4. the hair;
- 5. the nails.

THE SWEAT GLANDS

exist all over the skin. The gland starts as a coil in the dermis, and opens invisibly on the skin surface. The secretion is a clear watery fluid, 99–99·5 per cent water, also containing chlorides, lactic acid, urea nitrogen and other substances.

THE APOCRINE GLANDS

are large sweat glands, whose ducts open into hair follicles to which they are attached, and rarely on to the surface of the skin. They are coiled tubular glands, with a duct leading down to a coil of secretory tubules. They are found in the axillae, ano-genital areas, nipple and areola, but do not develop fully until puberty. Modified apocrine

glands occur in the external ear, producing wax; and on the eyelids, where cystic blockage may occur. The product of the glands is a whitish, sterile fluid, and contains proteins, carbohydrates and other substances. It is evoked in response to stress, pain, fright or sexual activity.

THE SEBACEOUS GLANDS

are found all over the body, except on the palms and soles. The glands have no lumen and their secretion is the result of decomposition of their cells, being discharged through the sebaceous duct into a pilo-sebaceous follicle. They are multi-lobulated, and appear as a pouch hanging on the outer side of the follicle. The secretion is known as sebum, and contains fatty acids, cholesterol and other substances.

THE HAIR

A hair consists of a root composed of non-keratinized cells, and a shaft composed of keratinized cells. The shaft extends from the skin surface to the free end of the hair. The root and its lower end are called the hair-bulb and contain the hair matrix cells. They comprise all the part below the skin surface, and lie in what is called the hair-follicle. A pointed projection of the dermis protrudes into the hair-bulb, and is called the papilla. It is luxuriantly supplied with nerves and blood vessels, and contains melanin which is responsible for the pigment of dark hair.

The distribution of the hair is universal except for the palms, soles, dorsal aspect of the distal phalanges of the hands and feet, the penis, the labia minora and the lips.

There are three types of hair:

1. downy or lanugo hairs which cover the face (except the beard and moustache areas), hands and limbs;
2. long soft hairs, which cover the scalp, beard, moustache, axillae, and pubes;
3. stiff hairs which are found on the eyebrows, eyelids, and in the nose and auditory meatus.

Hair growth varies according to the area. The average daily rate of scalp hair growth is 0.35 mm, and a hair may continue growing for from 2 to 6 years before falling. An average number of scalp hairs is

100,000, and the usual daily loss is 20 to 100. This is comforting consolation for those who imagine they are going to lose all their hair when they find a daily number on their brush or comb.

The reason why baldness does not occur as a result of this constant fall is due to the cyclical growth of hair. In every single hair follicle there is a resting period followed by a growth period; the loss of hair is not noticeable because neighbouring follicles have differently-timed cycles, and are at the same time at different phases of the cycle.

On other areas such as the trunk, eyebrows and limbs the growing period of the hairs lasts about 6 months, so that the hairs do not grow very long.

The influences on hair growth are complex and perplexing. The male hormone testosterone prescribes the sex hairs of men and women. It also provokes the growth of beard hair, yet its presence is a condition for the development of male scalp baldness. Eunuchs never become bald. Other hormones have little effect on hair growth except the pituitary which, through its connection with the adrenals and the gonads has an indirect influence.

The function of the hair is to protect the skin against minor harmful influences; for example, eyebrows direct sweat away from the eyes, and the nasal vibrissae filter air. It also acts as a thermoregulator, a promoter of sweat evaporation, is a sensitive tactile organ, and provides sexual attraction.

The health of the hair depends on the health of the individual. The visible hair is a dead structure, and no amount of singeing, brushing or oiling will alter its fundamental vitality, although its appearance may be improved.

NAILS

Nails are translucent, compact, solid plates of keratin. The matrix lies beneath the nail-fold. The paronychium is the soft tissue surrounding the nail border.

The average *growth rate* is 0.1 mm daily. A finger nail takes about 100–150 days to reproduce itself, and a toe nail about three times as long. Growth may be affected by disease, and nail shedding, splitting or ridging may accompany long illnesses, or malnutrition. In some cases, there is no obvious cause for such nail disorders. Nail-growth is increased by biting them, and like hair, their growth is accelerated in summer-time.

Their *function* is the indispensable one of picking up small objects. They are also bright ornaments to the fingers, and in disease may often indicate signs of disorders internally situated.

Dermis

The dermis may be divided into two parts:

1. the papillary;
2. the reticular.

The *papillary part* lies snugly against the epidermis above. The papillae strike up into it at irregular intervals, so that the alternating areas of epidermis which drop down produce the effect of a draped curtain. These epidermal drapes are called rete pegs.

Most of the papillae contain blood vessels, and some contain nerve elements, such as tactile corpuscles.

The *reticular part* contains connective tissue bundles, below which lies the subcutaneous tissue.

The dermis contains the following structures:

- (a) connective tissue fibres;
- (b) cellular elements;
- (c) blood vessels;
- (d) nerves;
- (e) muscles;
- (f) lymphatics.

CONNECTIVE TISSUE FIBRES

There are three varieties: collagenous, elastic and reticulin.

Collagen fibres form 95 per cent of the total. Collagen is an albuminoid substance of which the bundles are comprised. These present a wavy appearance, and are held together by a ground substance. Fibroblasts lie between the bundles.

Elastic fibres run parallel or obliquely to the collagen, and enclose the bundles.

Reticulin fibres probably ensure stability between dermis and epidermis.

CELLULAR ELEMENTS

A. In health

1. Migratory cells

(a) Leucocytes lie sparsely around blood vessels and lymphatics.

(b) Histiocytes resemble fibroblasts, having large round or oval kidney-shaped nuclei. They are also called reticulin cells. Their function is to absorb specific material, and form reticulin fibres. Under certain conditions they may change into epithelialized cells (see below).

2. Fixed cells

(a) fibroblasts are spindle-shaped with elongated nuclei.

(b) mast cells are histiocytic and spindle-shaped with an oval or round nucleus. They produce heparin and histamine. Normal skin contains few of them.

B. In disease

Apart from polymorphonuclear leucocytes, and lymphocytes, which play their accustomed roles as seen in other inflammatory diseases, other cells sometimes play a diagnostic part in skin disorders.

Eosinophils are significant in dermatitis herpetiformis.

Macrophages are phagocytizing histiocytes, and when fused, appear as multi-nucleated foreign body giant cells. As such, they particularly appear in gout.

Epithelioid cells are altered histiocytes and together may form Langhans giant cells, and as such appear in tuberculous, sarcoidal and syphilitic lesions.

Plasma cells occur in most chronic inflammatory conditions.

Foam cells are histiocytes ingesting lipoids, and are easily identified in the lesions of xanthoma.

BLOOD VESSELS

A capillary plexus exists between the dermis and the subcutaneous tissue, and in the sub-papillary area. Always varying, its position can never be accurately defined. The deep vessels have three layers of cells, the superficial vessels have one.

The glomus consists of an arterial segment, the Sucquet-Hoyer canal, and a venous segment, and is found on the tips of the fingers, toes, and under the nails. It is a local temperature regulator.

NERVES

The skin is supplied from non-medullated and medullated fibres, which reach it from bundles in the subcutaneous tissue.

The sensations of touch, spatial discrimination, and temperature on hairless skin such as the palms, soles, lips, nipples, penis and clitoris, have until recently been thought of as being mediated by specialized end-organs, named Meissner's tactile bodies, Pacinian corpuscles, Krause's end bulbs, or Merkel-Ranvier discs. This conception is now in doubt, and such sensations are considered nowadays to be mediated through the free sensory nerve endings in the epidermis.

MUSCLES

Smooth or involuntary muscle is found all over the skin, except on the neck, and in the facial muscles of expression. Smooth muscle is attached to the hair follicles, as arrectores pilorum, the tunica dartos of the scrotum, and the fibres of the areola of the nipple.

LYMPHATIC VESSELS

Few exist, and in the dermis present a spongy network, passing to deeper and larger plexuses in the subcutaneous tissue.

SKIN SURFACE LIPIDS

These cover the surface with a watery greasy film, called sebum, whose usefulness is debatable.

KERATIN

This is a fibrous protein, found in the horny layer of the epidermis, hair and nails.

WATER

The skin contains 18–20 per cent of the total water-content of the body. There is continuous invisible evaporation from the surface.

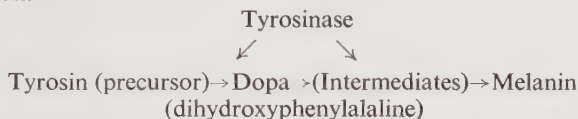
PIGMENTATION

The colour of normal skin originates from:

1. melanin;
2. oxyhaemoglobin;

3. reduced haemoglobin;
4. carotene;
5. melanoid.

Melanin results from the enzymatic oxidation of tyrosine by tyrosinase, which is attached to the melanocytes at the epidermo-dermal junction.



ABSORPTION

This occurs to a considerable degree, and the skin's absorptive capacity is greatly increased by abrasion. Hormones can be rubbed in, and it has been shown that iodine sprayed on the skin may be detected in the urine 20 minutes later. It has also been proved that water may be absorbed.

SENSATION

There are four types of sensation: pain, touch, cold and warmth, which are distinguished objectively and subjectively.

1. Pain sense. Pain may be caused by physical, chemical or mechanical irritation.

2. Touch sense. Touch spots are irregularly placed and are smaller where discrimination is acute. Touch stimuli are received from hair follicles, and the intervening skin.

3. Itching sense. This arises from terminal nerve-endings close to the skin surface (itching does not occur when the epidermis is absent).

4. Temperature sense. This sense is probably mediated through the free sensory nerve endings in the epidermis.

PATHOLOGY

The pathological changes found in skin diseases are often not diagnostic. The pathologist has to rely to a great extent on the clinical information supplied to him, which frequently is too trivial to be of much assistance. It should be a cardinal rule that a

good description of the case be sent with the specimen. Even so, in many cases, the pathologist may only state that the findings are compatible with the clinical ones, such is the close histological resemblance of some different skin diseases. In some conditions, however, his report reveals or confirms the diagnosis.

The terminology of the dermatological pathologist contains some words peculiar to the specialty, and others which are used generally in pathology. It is essential to know some of the former, so that the more obvious minutiae of histological sections may be understood.

The following are the commonest changes in the epidermis.

1. Hyperkeratosis, which is hypertrophy of the horny layer. This is most classically seen in a corn.

2. Parakeratosis is the retention of nuclei in the cells of the horny layer. It is well seen in psoriasis, and other scaly conditions.

3. Acanthosis is an increase in the depth of the prickle-cell layer. It occurs in psoriasis, warts, and other conditions.

4. Spongiosis is inter-cellular oedema, a part of the picture of dermatitis and eczema.

5. Acantholysis is a detachment of epidermal cells from each other, which produces clefts, vesicles and bullae in the epidermis. It occurs in the bullous condition called pemphigus.

The following are the commonest changes in the dermis.

1. Hypertrophy or atrophy of fibrous tissue, as in a keloid, or senile skin, respectively.

2. Capillary changes, as in lupus erythematosus, or scleroderma.

3. Collagen degeneration, as in senile skin, or scleroderma.

CHAPTER 2

History, Examination and Diagnosis

The traditional approach to a patient with a skin disease has usually, in the past, been objective. That is to say, examination and description of the lesion preceded any investigation into the history of the patient's condition. Almost all skin diseases generally can be diagnosed in this manner, because all the signs necessary to make a diagnosis are immediately visible, with a few exceptions. It is a good exercise, in the making of a diagnosis, but too often this method causes one to abbreviate history taking, which has many pitfalls. It also reduces the doctor-patient relationship from the start; for good rapport between them is often essential to effective treatment.

The recognition of the various types of lesions which may occur is the first essential towards making a diagnosis. This is sometimes more difficult than it sounds. But before this is done the patient should be put at ease, whilst all the senses must be alerted to pick up clues to the diagnosis as the patient approaches the doctor, just as in the diagnosis of a disorder of any other system. The author, in fact, believes that a subjective approach is more rewarding than the objective one. The patient who has pruritus ani, or a submammary rash, or lesions elsewhere on a clothed area, be it chest, abdomen, back or groin, should be questioned generally, so that an idea of the evolution and the symptomatology of the disease can be acquired. The patient will also be more co-operative and relaxed, and equanimity will be maintained, before his being submitted, naked if necessary, to the analytical gaze of the physician. Some patients are very proud and anxious to display themselves, while others are shy and timorous; a subjective approach is more remunerative, and allows one to be more in control of the situation, and therefore better able to construct the diagnosis.

Exceptions to this rule of course exist, especially when the face, hands, scalp or legs are involved. In many cases, a diagnosis can be mentally made at a glance and confirmed by the history obtained. In the case of warts, which unfortunately comprise so large a proportion of all cases, the diagnosis and questioning are simple and brief.

Nevertheless, different physicians have different methods of approach in the diagnosis of a skin disease. Some prefer to investigate the entire history of the patient before examination; others prefer to see, if possible, a sample of the disease, so that what seem to be pertinent questions can be asked at first. Both methods in experienced hands usually reach the same result.

Whichever method is applied, the skin disease must not be treated separately from the patient, for treating the part will never cure the whole.

In skin disease, as in diseases of any other system, it is essential to keep an open mind about the diagnosis during the history and examination. In a weak moment, one may be unintentionally misguided by a doctor's letter accompanying the patient, or by the patient's idea of the diagnosis. Sometimes the dermatologist may be confronted by a patient who speaks of his troublesome wart, and this statement may be confirmed by his doctor, who owing to a cursory examination, or bad light, has failed to note that the lesion is a corn. The wrong treatment for this condition, as for many others, can lead to unnecessary complications, and delay in healing.

The diagnosis of any disease is an exciting exercise in the use of deductive powers, not least where skin diseases are concerned. It is also a relief to the patient for him to realize that his ailment has been accurately diagnosed.

A working classification of common disorders, a knowledge of gross lesions and their distribution, combined with constructive history taking, should result in the correct diagnosis in all but uncommon diseases. For the latter, wide experience and supplementary aids, both histological and biochemical, will be required to discover the answer.

History Taking

A good history is often essential, particularly in chronic cases. This includes name, address, age, marital state, number of children, and occupation. Some of these factors may lead to a preliminary clue.

The following questions should be asked, although many may be excluded, if one is confident about the diagnosis, either as a result of some pointed answers as the patient is being questioned, or because one has seen and recognized the lesions. A complete history should include the following questions, but, as has been said, many will be irrelevant in some cases, and with experience, the pertinent ones to ask will be obvious.

PRESENT COMPLAINT

Where did it start?

Did it spread?

Does it come and go?

Is it wet, or dry?

Does it itch?

TIME OF ONSET

The following conditions are neither inclusive, nor exclusive.

Infancy: subject to atopic dermatitis or infantile eczema

naevi

pyogenic infections

Childhood: subject to warts

ringworm

urticaria

alopecia areata

Adolescence: subject to acne

Middle age: subject to neurodermatoses and tumours.

Old age: subject to tumours, benign and malignant.

PERSONAL HISTORY

How does the patient sleep? Insomnia is usually a reflection of tension.

Do any foods or liquids make the rash worse? Chocolate often worsens acne; alcohol, hot liquids and coffee and tea may make rosacea worse.

Are the periods regular, and does their occurrence upset the rash? Both factors may influence acne and rosacea.

What cosmetics does the patient use?

What hobbies has the patient? The answer may reveal a cause of contact dermatitis.

What is the patient's job?

Has the rash any relationship to seasons? Acne, psoriasis and ichthyosis are less troublesome in summer, pityriasis rosea is common in spring and autumn, and plant dermatitis in spring and summer.

FAMILY HISTORY

This is important when any of the conditions in chapter 20 are being considered, or alopecia, atopic eczema, psoriasis, rosacea, seborrhoeic dermatitis, and urticaria.

When infective conditions such as impetigo, pediculosis, or scabies are present, contacts must be known.

The next hurdle is *the identification of lesions*. One of the most important factors for this examination is good light. Many misdiagnoses are made because the patient casts a shadow on the rash or the light is not powerful enough. Facilities must also exist for complete examination of the patient if necessary.

The lesions of skin diseases may be primary or secondary.

PRIMARY LESIONS ARE

Macules

These are flat, circumscribed, discoloured lesions, of varying shapes and sizes, not raised above the skin.

Papules

These are raised, firm circumscribed lesions up to a centimetre in size.

Wheals

This is a circumscribed type of elevation associated with itching or tingling.

Nodules

These have the same characteristics as papules but are larger, and usually can be felt to lie more deeply in the skin.

Tumours

These are larger than nodules, and may be elevated or very deeply placed in the skin.

Vesicles

These are well-defined small collections of fluid.

Bullae

These are large vesicles.

Pustules

These are circumscribed elevations of free purulent fluid. In some diseases, pustules may be sterile, as in acne.

SECONDARY LESIONS

Scales

These are dry or greasy masses of dead tissue from the horny layer. They may be dry and silvery, as in psoriasis, or greasy and yellowish, as in seborrhoeic dermatitis.

Crusts

Crusts must be carefully differentiated from scales, and the novice often confuses them. Crusts are masses of dried exudate, bacteria and leucocytes. They are a dirty yellow colour, and may be soft, dry and friable, or thick and hard. The surface of a crust is more uneven than that of a scale. They are not composed of well-defined layers, as scales are, and their general consistency is more lumpy.

Ulcers

These are irregularly-shaped excavations, resulting from necrosis of tissue including complete loss of epidermis and dermis. Each ulcer has a shape, floor, base, edge and secretion. All ulcers leave a scar when they heal.

Scars

Scars are the result of damage to the dermis.

Two peculiarities associated with secondary lesions need to be described. They are:

1. Koebner's phenomenon.
2. Nikolsky's sign.

Koebner's phenomenon is the appearance in a patient, usually with psoriasis or lichen planus, of features of the disease in an area of skin that has been traumatized. For example, a patient with psoriasis is scratched by a thorn or nail; within a short time, psoriasis may appear in the injured area. These are the commonest conditions giving rise to this phenomenon, although plane warts may occasionally be liable to do so.

Nikolsky's sign is a sign elicited in pemphigus and describes the facility with which an apparently normal epidermis can be separated from the dermis by pinching or rubbing it. This is due to epidermal vacuolation.

A summary of the different types of lesions and some of the conditions associated with them

MACULES

Measles	Neurofibromata (café-au-lait spots)
Freckles	Vitiligo
Naevus flammeus	Addison's disease
Drug eruptions	

SCALY MACULES

Tinea corporis	Seborrhoeic dermatitis
Pityriasis rosea	Tinea versicolor

PAPULES

Acne	Melanoma
Warts	Basal-cell carcinoma (rodent ulcer)
Pigmented naevi	Molluscum contagiosum
Xanthomata	Tuberculids
Granuloma pyogenicum	

SCALY PAPULES

Psoriasis	Contact dermatitis
Lichen planus	Localized neurodermatitis
Atopic dermatitis	Syphilis

WHEALS

Urticaria	Insect bites
-----------	--------------

NODULES

Warts	Molluscum sebaceum
Haemangiomata	Syphilis
Chilblains	Basal cell carcinoma
Erythema nodosum	Lipomata
Erythema induratum	Neurofibromata
Molluscum contagiosum	

VESICLES

Herpes zoster	Burns
Herpes simplex	Atopic dermatitis
Insect bites	Dermatitis herpetiformis
Chicken-pox	Scabies
Contact dermatitis	

BULLAE

Erythema multiforme	Impetigo
Pemphigus	Herpes zoster
Pemphigoid	Contact dermatitis (poison ivy)
Dermatitis herpetiformis	

PUSTULES

Folliculitis	Herpes zoster
Acne	Herpes simplex
Rosacea	Smallpox
Chicken-pox	

CRUSTS

Any vesicular or bullous dermatitis	Any ulcerating disease
--	------------------------

SCARS

Any ulcerating disease	
------------------------	--

ULCERS

Trauma	Basal cell carcinoma
Self-inflicted conditions; e.g. dermatitis artefacta	Squamous cell carcinoma
Bedsore	Tuberculosis
Venous stasis	Syphilitic gummata

HYPERPIGMENTATION

Neurofibromatosis

Addison's disease

DEPIGMENTATION

Vitiligo

Albinism

PLAQUES

Psoriasis

Lichen planus

Seborrhoeic dermatitis

Paget's disease of the nipple

Atopic dermatitis

Lupus erythematosus

HYPERKERATOSIS

Corns

Ichthyosis

VEGETATIVE

Warts

Condylomata

Squamous cell carcinoma

ATROPHY

Senile skin

Lupus erythematosus

Scleroderma

When the nature of the lesion has been established, its characteristics should be defined according to size, shape, surface and colour.

The next step is to discover the distribution of the rash. In some diseases, the diagnosis can be made from the distribution, and in others, it is of much assistance. The inference should not be drawn, however, that because a disease does not present itself in its common pattern of distribution, that the disease does not exist. For example, psoriasis is commonly found on the extensors, but occasionally it will present itself as a solitary lesion in the external ear; a basal cell carcinoma is commonest on the face, but occasionally occurs on the trunk. On the other hand, rosacea only attacks the flush areas of the face.

A discriminatory appraisal of the distribution should be made, and a diagnosis on distribution alone, hesitatingly given.

The regional distribution of common conditions is as follows, and as will be seen, many diseases may be found simultaneously in different areas.

SCALP

Seborrhoeic dermatitis

Psoriasis

Tinea

Alopecia

Sebaceous cysts

FACE

Acne

Rosacea

Impetigo

Infantile eczema

Seborrhoeic dermatitis

Contact dermatitis

Sebaceous cysts

Neoplasms

Lupus erythematosus

Seborrhoeic warts

EYELIDS

Contact dermatitis

Warts

Xanthelasma

Neoplasms

LIPS

Herpes simplex

Cheilitis

Contact dermatitis

Neoplasms

Leukoplakia

MOUTH

Aphthous stomatitis

Leukoplakia

Lichen planus

Neoplasms

Pemphigus

EARS

Seborrhoeic dermatitis

Contact dermatitis

Psoriasis

Lupus erythematosus

CHEST

Acne

Seborrhoeic dermatitis

Tinea versicolor

Psoriasis

Pityriasis rosea

Seborrhoeic warts

AXILLAE

Contact dermatitis

Seborrhoeic dermatitis

Boils

Tinea

ABDOMEN

Pityriasis rosea

Psoriasis

Urticaria

Seborrhoeic warts

Drug eruptions

BACK

Acne

Seborrhoeic dermatitis

Psoriasis

Pityriasis rosea

Seborrhoeic warts

ANO-GENITAL AREA

Pruritus

Seborrhoeic dermatitis

Intertrigo

Tinea cruris (in males)

Contact dermatitis

Psoriasis

Pediculosis pubis

Syphilis

HANDS

Contact dermatitis

Hyperidrosis

Dysidrosis

Warts

Scabies

Atopic dermatitis

ARMS

Psoriasis

Contact dermatitis

Lichen planus

LEGS

Contact dermatitis

Neurodermatitis

Varicose dermatitis

Purpura

Erythema nodosum

Insect bites

FEET

Tinea

Warts

Dysidrosis

Psoriasis

Contact dermatitis

Atopic dermatitis

Corns

Apart from these visible signs, there are in some conditions other aids to diagnosis.

1. The *patch test* is described on page 55, in connection with contact dermatitis.

2. *Laboratory tests* to exclude fungus or bacterial infection. Special studies, such as an examination of the blood for L.E. cells, in lupus erythematosus, and platelet abnormalities in purpuric conditions. Other more routine tests, such as blood counts, sedimentation rates, and urine analyses may have to be carried out.

3. *Biopsy* is required in some chronic dermatoses, in all pigmented lesions which have been excised (see pigmented naevi) and malignant conditions.

Biopsy

In most cases, it is best to select a fully developed lesion. In vesicular, bullous or pustular lesions, very early lesions are preferable.

Normal tissue should generally not be included unless a large specimen is taken by the technician for processing. Finally, it is important to include subcutaneous fat, as some conditions reflect diagnostic features there.

Under aseptic conditions, and local anaesthesia, the chosen piece is excised and the wound sutured, or a special biopsy punch may be used. The specimen is placed on a piece of paper, smoothed out and allowed to dry for a minute or so, then, with the paper attached, put in a bottle containing 10 per cent formol saline, for transport to the laboratory.

Prior to biopsy or other surgical procedures, such as curettage of warts, it is advisable to obtain written permission for the operation from the patient or relatives.

Having taken the history, and identified the lesions, it is well to have in mind a tentative classification of disease. The cause of many skin conditions is still obscure, so that there is still a great deal of overlapping, no matter what sort of classification is adopted. At first, many of the names of skin diseases sound strange, but familiarity with them soon dispels dismay.

The broadest classification may be broken down into diseases, (a) benign, and (b) malignant. Malignant skin diseases in patients under the age of 40 are uncommon.

A more useful broad general classification is to break diseases down into:

1. Inflammatory, scaly and infectious.
2. Granulomatous.
3. Malignant.

The first group includes those in chapters 6–14; the second group, lupus erythematosus and sarcoidosis, and the third the appropriate conditions described in chapter 20.

Many conditions are perforce excluded by this elementary classification, which is intended only to be a jumping-off point in the elucidation of the diagnosis. Some conditions, such as warts or acne, obviously do not require such an aid to diagnosis.

A sound diagnosis depends on acute observation of the lesions and an accurate history, culminating in a careful assessment of them both.

CHAPTER 3

Treatment

Dermatological treatment, in the widest sense of the term, is identical with the management of any other speciality.

It should consist of treatment of the patient and the disease. It is extremely easy to forget the patient in the course of one's zeal to identify a lesion, or because of lack of time, or because one considers the condition too trivial to take seriously. Some patients are apparently less affected by skin diseases than others, and one must discover how much they are affected, in order to judge the amount of attention they should receive, as opposed to the disease. Some patients, for example, show a sense of profound revulsion as owners of a wart, or (in the case of women) of downy hair on the upper lip, whilst others acquire a more balanced attitude. The treatment of these factors, and the patient's general condition, should be the *sine qua non* on which therapy is based. Successful end-results can be obtained in treating only the lesions, but it is far more satisfactory for both doctor (and patient) to treat both patient and disease.

It used to be said by the ignorant, even ten years ago, that patients with skin diseases were never cured, nor did they die. Both assertions were untrue. Within recent years this phrase has been seldom heard.

In the recent past, pemphigus, and systemic lupus erythematosus, were classified as fatal diseases. These conditions still occur but sufferers may now be kept in reasonably good health for long, and even indefinite, periods by steroid treatment.

In the past, many conditions were cured, and to-day there are many more as a result of the benefits obtained by steroids, and the slightly less dramatic results obtained by antibiotics.

Although these two forms of drugs have revolutionized the management of certain skin diseases, they should only be used with discrimination.

Probably the most important ingredient in the treatment of skin diseases, as in the treatment of the diseases of other systems, is *rest*. This is particularly applicable to acute inflammatory conditions.

Patients find it extremely difficult to believe that rest will be very beneficial as, except for the irritation and the appearance of the rash, they are not much physically handicapped. If they are told, however, that the tissue of the skin differs little in its reaction to disease and rest, from affections of, for example, the lungs or stomach, the necessity for rest may be more easily understood. They should also be told that rest very definitely reduces the amount of itching, which is so vulnerable to environmental changes, and, when in an inflamed condition, to the petty stresses of daily life.

In generalized inflammatory conditions, the patient must be put to bed, and no compromise, such as lying on a couch, should be considered. The patient should be told that he may be incapacitated for 4-6 weeks. The good effects of bed-rest are quickly visible. The only exception is that old people should be allowed to sit out of bed for longer periods daily. In their case the avoidance of the development of broncho-pneumonia and bed-sores easily outweighs the consideration that additional ambulation may prolong treatment.

Otherwise patients should be allowed to go to the toilet when necessary, and sit up for a little while every day.

Anti-boredom devices are essential for otherwise active patients, and good sisters and nurses are adept in this art.

Diet has little place in skin diseases, except perhaps for slimming for the obese with intertrigo, or leg ulcers. Some patients with acne find that pastries and other confections provoke the appearance of lesions. Some with rosacea find that alcohol and hot or spicy liquids do the same. Sensitivity to food is a rare cause of skin disease, but when it is, the offending item must be proscribed.

Treatment of the disease can be divided into:

- (a) prophylactic measures.
- (b) curative measures.

Prophylactic measures can be adopted in any particular disease by avoiding substances or conditions which are known to provoke an eruption.

Examples are the avoidance of sunlight when light-exposed areas are affected, as in lupus erythematosus; the restriction of shaving, when the beard area is infected; the avoidance of long periods of standing or sitting, when varicose dermatitis is present; or withdrawal from situations known to cause contact dermatitis.

CURATIVE MEASURES

are many. They may be:

- (a) local, or external;
- (b) general, or internal.

External Treatment

A good general rule is to apply wet dressings or lotions to oozing lesions, and creams, ointments or pastes to dry lesions. If an ointment is applied to an oozing lesion, the exudate will force it from the area being treated, thus nullifying any potential benefits it might possess. Medicated baths are also sometimes useful.

DRESSINGS

The most commonly used are:

1. Normal saline: one teaspoonful of salt to a pint of water. This is the simplest to use.
2. Potassium permanganate solution in water: 1/8,000.
3. Liquor aluminium acetate (Burow's solution): half a teaspoonful to a pint of water. This is also made up in convenient tablets which when absorbed in water produce a similar concentration: they are called Domeboro tablets.
4. Silver nitrate: $\frac{1}{8}$ — $\frac{1}{4}$ per cent solution.

Their *action* is antipruritic and cooling, and to soften crusts; and to maintain these effects, they must be kept damp.

Their *indication* is in acute inflammatory conditions, such as acute contact or atopic dermatitis, when vesicles, pustules, and oozing lesions exist.

The *method of application* is to soak layers of cotton or unstarched linen in the cool solution. The dressing is gently wrung out, applied

damp to the lesions, and kept in place by a hand towel. The outer layers of cotton or linen are constantly removed, dampened, and reapplied to maintain a moist dressing.

BATHS

The most commonly used are:

1. *Bran*, or oatmeal, for senile pruritus or exfoliative dermatitis. 240 g of bran or oatmeal is enclosed in a muslin bag, and tied under the hot tap as the water runs into the bath.

2. *Tar*, for psoriasis. Four tablespoonfuls of liquor picis carbonis are added to a bath, in which the patient should lie for at least ten minutes.

3. *Potassium permanganate*, 1/8,000 solution in water, is useful for itchy, infected or eczematous conditions of the hands or feet, although this has a tendency to produce a rather dry skin.

4. *Emulsifying bath*, for infants with eczema. Add a tablespoonful of emulsifying ointment B.P. to a bath of about 45 litres.

5. *Sodium bicarbonate* is useful for localized itchy conditions. 240–700 g are added for an adult bath of 130–180 litres of water.

POLYTHENE COVERINGS

These may be particularly useful in psoriasis, chronic eczema, or localized neurodermatitis.

Polythene lay-flat tubing is a seamless plastic tube sold in various widths and gauges to accommodate the arms, legs, or trunk.

The cream or ointment to be used (usually a steroid in these cases) is applied fairly liberally, the polythene is then put on and kept in place and made airtight around the ends with adhesive tape. The dressings are left untouched for 24 hours unless there is much discomfort from the heat and/or moisture which develop. Sometimes, it may only be possible for the patient to wear such a dressing at night.

Although such a regime is no more curative than others, it does appear to produce speedier recovery.

POWDERS

These have protective and absorptive properties, and may also be antipruritic or astringent. Starch, talcum, and zinc oxide are common

ingredients. They are useful for lesions in creases, such as the groins. They must be cautiously applied to weeping and raw surfaces.

Examples of basic powders are:

1. Zinc. Starch and Talc dusting-powder B.P.C. This contains one part of zinc oxide and starch to two of talc.
2. Zinc. and Salicylic Acid dusting-powder B.P.C.

An example of a medicated powder is:

1. Dicophane dusting-powder B.P.C. DDT dusting-powder, 10 per cent.

LOTIONS

These are solutions or suspensions in water, or spirit.

1. *Calamine lotion*

This contains Calamine (zinc carbonate), zinc oxide, and glycerin in distilled water. It is apt to be too drying but is useful for insect bites, or urticaria.

2. *Oily calamine lotion*

is used when the first type causes too much drying. It contains calamine with various oils in a solution of calcium hydroxide.

3. *Steroid lotions*

A great number of proprietary forms are now used. Half per cent strength is often as useful as 2 per cent. They are also made to incorporate an antibiotic, and are used in this manner when an infective element exists. Steroids in any form have become the most useful weapons in dealing with itchy dermatoses. Their use is greatly enhanced if their potential dangers are continually kept in mind, and their employment carefully chosen.

These lotions are best used when small areas are involved; when they are large, steroid sprays may be tried. They are most useful in pruritus ani and vulvae, contact, atopic and neuro-dermatitis.

The following are a few examples of proprietary brands of steroid lotions: with hydrocortisone (Cortril), with fludrocortisone (Florinef), with fluocinolone (Synalar) and with triamcinolone (Adcortyl-E).

4. *Lot. Potass. Sulphurat. c. Zinc. B.N.F.*

contains sulphurated potash and zinc sulphate in camphor water, and is one of the most useful local remedies for acne.

5. *Salicylic Acid Lotion B.P.C.*

This contains:

Salicylic acid	2 g
Castor oil	1 ml
Industrial methylated spirit to	100 ml

Labelling: Caution. This preparation is inflammable. Keep away from a naked flame.

OINTMENTS AND PASTES

Ointments consist of animal, mineral, or vegetable oils as bases, in which various substances are incorporated. Ointments may thus be made anti-pruritic, antiseptic, astringent, or whatever quality is required.

Pastes are mixtures of powders and ointments. They are thicker, more protective, and more adherent than ointments, and therefore more difficult to apply and remove. The use of ointments and pastes is restricted, creams being preferred whenever possible.

The following are used as bases for ointments:

- (a) White paraffin
- (b) Lanolin
- (c) Carbowax
- (d) Emulsifying ointment
- (e) Aquosum ointment.

The following pastes are in common use:

- (a) Zinc paste; contains zinc oxide and starch in soft white paraffin.
- (b) Lassar's paste; substantially the same as zinc paste, with the addition of salicylic acid,

Examples of medicated ointments and pastes are as follows:

- | | | |
|-------------------|------|-----|
| 1. Salicylic acid | 1 g | 3% |
| Benzoic acid | 2 g | 6% |
| Carbowax to | 30 g | 91% |

This is Whitfield's ointment, used chiefly for fungal infections of the feet.

- | | | |
|---------------------|------|-------|
| 2. Calamine | 5 g | 16.7% |
| White soft paraffin | 25 g | 83.3% |

This is calamine ointment, and is used for chronic dry itchy dermatoses.

- | | | |
|-------------------------|------|------|
| 3. Precipitated sulphur | 1 g | 3% |
| Liquor Picis Carbonis | 2 ml | 6% |
| Salicylic acid | 1 g | 3% |
| Emulsifying ointment to | 30 g | 100% |

This is an excellent scalp ointment for widespread seborrhoea or psoriasis, when the condition is not acute.

- | | | |
|--------------------------|------|------|
| 4. Liquor picis carbonis | 1 ml | 3% |
| Lassar's paste to | 30 g | 100% |

A useful paste for sporadic lesions of psoriasis.

5. Some proprietary antibiotic ointments:

tetracycline (Achromycin), chlortetracycline (Aureomycin), oxytetracycline (Terramycin), chloramphenicol (Chloromycetin), soframycin-gramacidin (Soframycin).

They are very useful in infected conditions such as impetigo, or folliculitis; the organism should be identified and its drug sensitivity solved before prescribing the appropriate ointment.

It should be noted that penicillin ointment or cream must never be used on the skin, because of the great risk of sensitization, which results in a dermatitis extremely difficult to cure. The same applies to sulphonamide and antihistamine ointments and creams. Neomycin and the chemically related Framycetin may also sensitize the skin, especially in eczematous subjects.

6. Hydrocortisone Ointment B.N.F. contains hydrocortisone in a suitable greasy base such as 10 per cent wool fat in white soft paraffin. The cream is much more widely used.

There are many proprietary brands of steroid ointments, as there are creams (see below). They contain 0·1 to 2·5 per cent of the active agent.

CREAMS

These are soft ointments, usually composed of bases containing a high proportion of water, liquid paraffin, or arachis oil.

Examples are:

1. Calamine Cream B.P.C. Used for inflammatory conditions.
2. Zinc Cream B.P.C. Zinc cream. B.P. Also for inflammatory conditions, or as a vehicle for tar or ichthammol.
3. Crem. Zinc. et Ol. Ricin. B.P. Zinc and castor oil. Used mainly for infants.
4. Hydrocortisone Cream B.P.C. 1 per cent in a water soluble base. There are many proprietary brands of steroid creams; for example, with hydrocortisone (Efcortelan, Genacort, Cortril), with triamcinolone (Lederkort, Adcortyl-E), with fluocinolone (Synalar), with betamethazone (Betnovate). Some of them incorporate antibiotics, e.g. Hydroderm contains neomycin sulphate and zinc bacitracin; Remiderm contains triamcinolone acetonide and halquinol. Neocortef contains neomycin and hydrocortisone; some incorporate tar, e.g. Tarcortin Carbocort, Cortipix, and some iodochlorhydroxyquinoline, Vioform-HC, Domeform-HC, while some contain both: e.g. COR-TAR-QUIN.

They may be very effective in the following conditions:

Atopic dermatitis: for relatively small areas.

Contact dermatitis: for small areas, but less useful than in atopic dermatitis.

Localized neurodermatitis.

Pruritus ani and vulvae.

Nummular eczema.

Psoriasis.

ANTIPRURITICS

The following are the best:

Steroids

Calamine

Tar

Ichthammol

ANTISEPTICS

The following are the best:

Potassium permanganate

Cetrimide (Cetavlon)

Eusol (contains calcium hypochlorite and boric acid)

Iodochlorhydroxyquinoline (Vioform)

PAINTS

These are occasionally used on areas where creases exist, such as the anogenital areas or between the toes.

1. Magenta Paint B.P.C.

This contains magenta, phenol, boric acid and resorcin amongst other substances.

It is useful as an antipruritic, and also for its drying effects on moist lesions in the anogenital area, and between the toes.

It deteriorates after one month, and also stains the clothing.

2. Coal Tar Paint B.P.C.

Crude coal tar 10 g

Acetone

Benzene of, equal volumes to 100 ml

It is used predominantly in lichenified dermatitis and psoriasis.

3. Podophyllin Compound Paint B.P.C.

10–20 per cent podophyllin in Tinct. Benz. Co. is useful for anogenital warts, and plantar warts.

This paint is very irritating to the eyes.

Table 1 shows the quantities of preparations it is advisable to prescribe.

TABLE 1

	<i>Creams and Ointments (g)</i>	<i>Lotions (ml)</i>
Face	20	100
Hands	50	250
Scalp	50	250
Arms and legs	100	250
Body	200	500
Groins and genitalia	20	100
Dusting Powders	50 or 100 g	
Paints	10 or 20 ml	

Internal Treatment

The number of drugs which when given internally benefit dermatoses is small, but the number of dermatoses which are benefited is large in comparison.

The drugs are:

1. Steroids
2. Antibiotics
3. Griseofulvin
4. Antihistamines
5. Anti-malarials
6. Endocrines
7. Vitamins
8. Sedatives

1. STEROIDS

The discovery of cortisone and ACTH has revolutionized the treatment of many dermatoses, as it has done in the management of conditions previously unmanageable in other branches of medicine. As should be known, they are double-edged weapons, capable of doing as much harm as good. In certain dermatoses, such as pemphigus, and systemic lupus erythematosus, their life-saving, or life-prolonging qualities, far outweigh any consideration of the harm they can do. In many non-fatal conditions, however, in which they produce remarkable improvements, there may be a tendency to prescribe them without duly weighing the pros and cons. This consideration is absolutely essential before their administration is begun.

In benign conditions, the following points should be considered: (1) are steroids the most effective treatment? (2) for how long must they be given?

The recognized contra-indications to the giving of steroids are:

1. Chronic nephritis
2. Cardiac failure
3. Peptic ulcer
4. Coronary thrombosis of recent origin
5. Diabetes mellitus
6. Senile osteoporosis

The following conditions are those in which steroids have been found to be useful, and may be given after due consideration:

1. Atopic dermatitis
2. Contact dermatitis
3. Erythema nodosum
4. Exfoliative dermatitis
5. Lichen planus
6. Lupus erythematosus: (a) systemic, (b) localized
7. Pemphigus
8. Psoriasis; in extensive and disabling cases
9. Urticaria; when acute, a short course may be justified

Steroids may also be given intradermally, as into the localized lesions of lupus erythematosus, or hypertrophic scars.

Steroids in ointments and lotions have already been dealt with (*vide supra*).

The drugs employed parenterally are:

1. Prednisolone, given by mouth in tablet form;
2. ACTH, given intravenously or intramuscularly.

Cortisone is not now used in dermatology, and has been replaced by prednisolone and other steroids such as triamcinolone and methylprednisolone.

2. ANTIBIOTICS

(broad-spectrum) such as tetracycline (Achromycin) or oxytetracycline (Terramycin) or erythromycin. They are given in tablet or capsule form. They may be used in:

- Acne.
- Boils.
- Carbuncles.

Inflammatory dermatoses complicated by infection.

Although acne lesions are sterile, the tetracycline group of antibiotics often produce the most satisfying results, for reasons unknown; and relatively small doses can be given for long periods (see p. 191).

3. GRISEOFULVIN

This is an antibiotic obtained by the fermentation of several species of penicillia.

It is extremely effective in most types of superficial fungus disease; in fact, against all known species of *Microsporum*, *Trichophyton* and *Epidermophyton*. It is not effective against *Candida albicans*. The antibiotic is fungistatic, and not fungicidal.

Its greatest value is in ringworm of the scalp and chronic infections of the skin and nails.

The drug is given in 250 mg tablets, 3 or 4 times a day, or the same dose all at once. Recent preparations permit the giving of lower doses, which are equally effective. Treatment in chronic nail infections may have to last a year or more. Severe reactions are rare, but reversible side-effects such as diarrhoea, headache, leucopenia or albuminuria may occur.

4. ANTIHISTAMINES

These may be given by mouth in tablets of varying strengths, 3 times daily, according to the proprietary tablet. Examples are: triprolidine hydrochloride (Actidil) 2.5 mg, and chlorpheniramine maleate (Piriton) 4 mg.

They are used predominantly in urticaria.

They must not be given topically, as the sensitization rate is high.

5. ANTI-MALARIALS

Nivaquine (Chloroquine Sulphate). This is sometimes effective in chronic lupus erythematosus. 200 mg tablets twice daily is the usual initial dose, which is later reduced.

Toxic symptoms are not uncommon, and vary from pruritus and diarrhoea to serious ocular complications such as amblyopia (see treatment of chronic lupus erythematosus).

6. ENDOCRINES

Oestrogens and progesterones by mouth are sometimes used in acne, and occasionally effective.

7. VITAMINS

Vitamin A is sometimes useful in ichthyosis. Otherwise vitamins are given for general rather than specific effects.

8. SEDATIVES

These may be essential to allay itching, particularly at night, when the work of a day's treatment may be undone, although daytime sedation is also sometimes required.

For daytime, give phenobarbitone 15 mg three times a day, or Drinamyl (amphetamine and amylobarbitone) or Anxine (amphetamine, amylobarbitone and mephenesin, a muscle relaxant).

For night time, give Tuinal 200 mg or Nembutal 200 mg.

Apart from the measures described above physical agents (see, for example, Fig. 2) are also used in the therapy of skin diseases.

They are:

diathermy

cautery

carbon dioxide

ultra-violet rays

X-rays

Grenz rays

thorium X

dermabrasion



FIG. 2. Curettes, for the removal of warts.

CAUTERY AND DIATHERMY

With both these methods an electrified needle destroys the lesion with which it is in contact (Fig. 3).

Both methods are used for destroying common warts, seborrhoeic warts, senile keratoses, and certain naevi.

CARBON DIOXIDE

This may be used as (a) a solid stick and (b) in the form of a slush, which is obtained when the stick is mixed with acetone.

(a) In the form of a stick, its indications are in the treatment of plantar warts, seborrhoeic warts, senile keratoses.

(b) In the form of slush: patches of chronic lupus erythematosus, and chronic exuberant acne lesions.



FIG. 3. Cautery machine, handle and point.

ULTRA-VIOLET RAYS

Indications are: acne and psoriasis.

Contra-indications are: lupus erythematosus, photo-sensitive eruptions, and a history of pulmonary tuberculosis.

X-RAYS

X-rays are produced in a tube made of glass, containing two electrodes, an anode and cathode. The cathode is heated by a filament and produces electrons, which are attracted to the anode by applying a high voltage between anode and cathode. The energy at the anode is transformed into heat; the higher the voltage, the more penetrating the radiation.

The usefulness of X-rays is declining, and cases must be carefully selected.

Indications are:

- acne, in some carefully selected cases
- basal cell carcinoma
- circumscribed neurodermatitis
- otitis externa
- seborrhoeic dermatitis, when localized to intertriginous areas

GRENZ RAYS

These are less penetrating than X-rays, and therefore potentially less dangerous.

They are used for:

- lichen planus
- psoriasis
- circumscribed neurodermatitis

THORIUM X

This is a radio-active element, which is prepared in alcohol or varnish, so that it can be painted on the affected area.

Indication for its use are:

- alopecia areata
- mycosis fungoides, early phase

Its efficiency is very questionable, and its future in the balance.

DERMABRASION

The skin is abraded with a device similar to a dentist's drill, the drill being replaced by a wire-brush mandrel.

Indications are:

- acne scars
- accidental tattoos, e.g. coal dust in miners

Its use is very limited and with acne scars is not entirely satisfactory, even in the best hands.

It will be observed from the foregoing pages of this chapter, that whilst most skin diseases are curable or controllable, the exact mechanism of the drug used is commonly obscure. Why sulphur is

topically useful in acne and seborrhoea, and steroids in pemphigus, for example, is not understood. The use of most drugs in dermatology is empiric, that is, treatment is based on the results of experience, rather than scientific knowledge. If the contra-indications and the complications of drug therapy are known, however, the experienced physician should have little difficulty in the management of this aspect of skin diseases.

CHAPTER 4

Eczema and Dermatitis

More than half of all skin diseases fall under this heading. Both terms denote an acute, subacute, or chronic inflammatory condition, characterized by

1. Erythema and oedema;
2. Discrete or grouped vesicles, changing to weeping and crusted lesions, and/or papules and scaling;
3. Itching or burning, with scratching or rubbing, which later leads to lichenification of the skin.

Both terms, eczema and dermatitis, are used synonymously.

The cause of eczema or dermatitis is, on the one hand, external or traumatic, and, on the other hand, internal or constitutional. When external causes are responsible the condition is called contact dermatitis. When internal causes are responsible, four principal varieties of eczema or dermatitis may be produced:

1. Atopic eczema
2. Nummular eczema
3. Pompholyx
4. Seborrhoeic eczema (p. 71)

Atopic Eczema

*Atopic Dermatitis; Allergic Eczema; Besnier's Prurigo;
Disseminated Neurodermatitis*

Atopy is a term used to describe the following phenomena:

1. A marked familial tendency to allergic diseases such as asthma, hay-fever, urticaria, and rhinitis.

2. A high degree of hypersensitivity to protein substances.
3. Nervous system disturbances reflected by unusual reactions to heat, cold, and emotional tensions.

Atopic eczema is frequently associated with these phenomena. The atopy is separate from the eczema, as can be shown by the fact that if it transpires that the patient is sensitive to a certain type of food, the giving of it, or its deprivation, will not affect the eczema one way or another.

PATHOLOGY

The acute phase is characterized by oedema in the epidermis. Varying degrees of acanthosis also occur. As the condition progresses and becomes more chronic, hyperkeratosis and parakeratosis follow. The dermis invariably shows a perivascular infiltrate. The changes are non-specific.

CLINICAL FEATURES

These vary with the age of the patient, and occur as three varieties.

1. *Infantile Eczema*

The onset is usually about the third month. The skin is red, with small vesicles on a puffy surface, and small cracks ooze serum (Fig. 4). The sites commonly attacked are the face, except for the skin around the mouth, nose, and eyes; the forearms, wrists, outer parts of the legs, and the flexures. Recovery usually occurs between the third and fourth year after periods of remissions and relapses, which is a period of great trial for the parents and child.

A non-atopic variety of infantile eczema occasionally occurs, which responds to treatment, and does not last more than a few weeks. If it does do so, it shows itself to be of the atopic type.

2. *Childhood Type*

This may follow directly from the infantile variety, arise as a fresh outbreak later, or appear for the first time. Papular and lichenoid lesions are commonest, and the flexor surfaces are those most often affected. It usually disappears by the age of 10 or 12.

3. *Adult Type*

This may or may not be preceded by a history of infantile eczema, and/or the childhood type. The lesions are papular and lichenified,

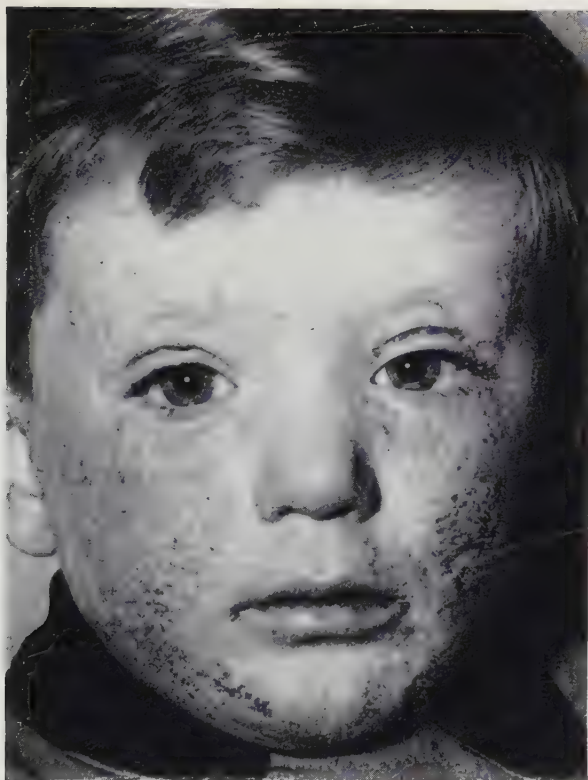


FIG. 4. Infantile eczema. Crusting and scaling, with typically relative freedom of the circumoral area. The worried expression is also typical.

and excoriations are common. Crusting and exudation follow scratching (Fig. 5).

The sites commonly attacked are the flexures, and then the front and sides of the neck, eyelids, forehead, face, chest, wrists, and the backs of the feet and the hands. In mild cases, only one or two areas

may be affected. The course is intermittent and there may be relatively long periods of freedom from lesions, although chronicity is the rule.



FIG. 5. Eczema of the nipple. Weeping lesions without induration or infiltration which occur in Paget's disease of the nipple.

Other Findings

Itching is severe, and a change in room temperature, a change in mood, or friction from wool is enough to irritate the skin.

Sweat retention is often marked, *Chronic secondary bacterial infection* is common.

A tense or aggressive *mental attitude* is often noted.

No young patient with this disease must be vaccinated because of the danger of eczema vaccinatum which in its severest form

causes death. Adults must be carefully assessed before being vaccinated.

DIAGNOSIS

This is made by the character and distribution of the lesions, the itching, and the family and allergic history.

The disease must be distinguished from *contact dermatitis* in which itching is inconstant and the lesions are relatively localized, any area being affected. In *seborrhoeic dermatitis* the lesions are not particularly itchy and are not on flexor surfaces. *Localized neurodermatitis* is characterized by well-circumscribed, occasional and itchy lesions on any area.

TREATMENT

This condition calls for more skill and patience on the part of the physician than any other skin disease.

Sympathy and understanding are an enormous help to the patient (and parents), and even an intimation of pessimism or despair may have disastrous consequences. It is important that the patient or parents should have an idea of what is at the basis of the disease.

There is no specific therapy but a great deal can be done to help these difficult cases.

Prophylactic measures

Sudden changes of temperature must be avoided, such as undressing in a cold atmosphere or entering too hot a bath. In summer, light clothing must be worn at night and a well-ventilated room is essential. Irritating clothing, such as woollens, or sharp straps, or buckles attached to clothing, must be avoided. Soaps, detergents, and other household cleansers must be avoided as much as possible.

Pillows and mattresses should be made of foam rubber, and regular hours of sleep (and meals) are advisable.

Curative measures

Nothing must be applied which will irritate the skin or overheat it. For moist lesions, lotions should be used; for dry ones, creams or ointments.

For the *acute* or moist forms of the disease (or any dermatitis) Burow's solution (aluminium acetate), calamine lotion, or a steroid

lotion or spray may be used, but the effect of over-drying the skin must be avoided, since it produces fissures; if they occur, creams should be applied. Emulsifying baths are very useful in this acute phase (see p. 26).

For the *chronic* or dry forms of the disease, applications of steroid creams are useful, and when extra penetrative effect is required they may be applied under a polythene covering; steroid intoxication can occur with this technique. Sometimes these preparations fail to relieve or improve the condition, and other ointments, such as 2 per cent liquor picis carbonis or ichthammol in zinc ointment, may be used.

Antihistamine preparations must not be applied (see p. 34).

Systemic therapy in the form of steroids may be given only in severe cases after much thought. Although the improvement is dramatic the chief drawback is that when the drug is withdrawn, inflammation may recur in a form more furious than before, and therefore the difficulties of stopping the drug, or restraining the patient from obtaining it from other sources, are very great. It should only be used for extremely or extensively involved, and disabled patients. It should not be given to children.

Sedation by barbiturates at night may be given to reduce night scratching. Antihistamines by mouth, in the form of triprolidine (actidil) 2.5 mg t.d.s., are sometimes helpful in reducing pruritus. For children, graduated doses are given.

Psychotherapy as a supportive measure in the treatment of atopic dermatitis is important, although it should not be one of the main methods of treatment. When confidential rapport between doctor and patient has been established, conditions of stress at home or at work should be discussed, and an optimistic prognosis put forward. Moreover, in the case of a sick child, the parents should be advised to refrain from acts of unconscious disgust towards the disease, in the form of acute anxiety to get rid of their child's disfigurement, or discussing the handicap he or she has to endure, especially in the child's hearing. Treatment by a psychiatrist is rarely if ever indicated, and such aid is generally not of much help.

Environmental change to hospital, or the home of a relative or friend, invariably produces a turn for the better, and this may reflect vague but real problems encountered in the home situation.

The overall aim in treatment, in fact, is to soothe by a combination of suitable applications, the avoidance of physical and/or psychological stress, and the indoctrination of hope and confidence in the outcome, as opposed to the destructive effects of melancholy.

Nummular Eczema (Discoid Eczema)

This chronic inflammatory condition is characterized by coin-shaped or nummular lesions, which may be dry or wet.

CAUSE

This is unknown. It occurs usually in tense middle-aged individuals.

PATHOLOGY

The picture is one of acute or chronic dermatitis.

CLINICAL SIGNS

Itching is common, and sometimes intense. The onset is insidious, and at first there are only one or two lesions, but they multiply. The lesions are papular, and consist of very small vesicles, which soon burst, leaving crusts. The lesions usually coalesce to form plaques. Any site may be affected, but commonly the back of the hands and forearms, the front of the thighs and the calves of the legs.

COURSE

This is difficult to predict, some cases clearing within a month or two, others following an indolent course of remissions and relapses for a year or more.

DIAGNOSIS

This disorder must be distinguished from *ringworm* which has a red margin with scaling, while the centre shows healing. In *psoriasis* there are no vesicles, and itching is absent.

TREATMENT

The same topical and internal measures are used as for atopic dermatitis.

Natural sunlight is very useful, while cold weather and too many baths must be avoided.

Pompholyx

Dysidrosis

This is an acute or subacute disorder of the hands (then named cheiropompholyx) and/or the feet (pedopompholyx), characterized by deep-seated itchy vesicles. Pompholyx can be considered to be an unusual type of eczema or dermatitis.

Pompholyx may be associated with excessive sweating of the affected areas, with emotional upsets, tinea of the feet, or occasionally with contact dermatitis.

PATHOLOGY

The vesicles are intra-epidermal, and commonly independent of the sweat-duct. There are few or no inflammatory changes to be seen.

CLINICAL SIGNS

Itching and burning sensations are usual, and occur most severely as a new lesion is being formed. The onset may be gradual or sudden. Lesions are vesicular, similar to boiled sago grains. The surface is at first smooth, but later, when the vesicle has ruptured, a brownish scale develops. The colour of the lesions is faintly white owing to their contents, and the surrounding skin usually shows little change. Sometimes this renders them difficult to see.

Sites: the sides of the fingers, the palms, and the soles are attacked. In severe cases all areas may be involved, in mild ones perhaps only a few lesions will be found on the fingers. The distribution is invariably bilateral. The nails in chronic cases are deformed.

DIAGNOSIS

The itching and character of the lesions are unmistakable, but the most important task in these cases is to discover the cause.

TREATMENT

If the cause is treated, the lesions clear up. But all too often it remains hidden, and symptomatic and empirical treatments have to be used.

External

Potassium permanganate 1/8,000 warm hand baths b.d. for 10 minutes each, followed by the application of zinc or steroid cream are most useful. X-rays may help considerably in chronic cases.

Internal

Oral steroids may have to be used in very chronic cases.

PROGNOSIS

Recurrences are not uncommon. The interval between them varies between months and years.

Contact Dermatitis

Dermatitis venenata, occupational or industrial dermatitis

This condition is characterized by redness, oedema, vesicles, and sometimes bullae, and a variable amount of itching. It is caused by chemical or vegetable substances coming into contact with the skin.

About 10 per cent of dermatological patients attending hospital suffer from this disease, although of course this varies in different regions and may well be higher in industrial areas and lower in rural ones.

CAUSE

Apart from the actual provoking cause, predisposing factors are present. They may be an individual susceptibility, or other conditions such as minor trauma, excessive sweating, atopic dermatitis or ichthyosis. Any of these in the mildest form can also be provocative agents.

The actual cause may be a *primary irritant*, or a *sensitizer*. A *primary irritant* is a substance, such as lime or nitric acid, which will produce inflammation on first contact with the skin, if permitted to act in sufficient intensity or quantity for a sufficient time. The rash produced by primary irritants is self-limited, and disappears reasonably rapidly. Prevention of a recurrence is easy, once the irritant is recognized, by means of special clothing, washing after exposure, and barrier creams.

Sensitizers, such as flour, or dyes, are a much larger problem. The number of exposures to the substance which will cause a rash

varies from a few to many. One baker, for example, will suddenly become sensitized to flour after handling it for years; another, following only a few exposures. This time factor varies incomprehensibly. Prevention in most cases is impossible, and complete avoidance of contact with the offending substance is the only safeguard.

Identification of the cause requires investigation of the patient's occupation, hobbies, household duties, wearing apparel, cosmetic articles, and holidays. The questions, though apparently exhaustive, may be fruitless. A daily diary may have to be kept by the patient, and in some cases, the work-bench or home surroundings visited by the physician.

Difficulties to be encountered may be gauged by the case of a woman who developed primula sensitivity in a friend's house, even though the plant was concealed behind a screen, or that of a clerk who worked for a market gardener and developed tomato sensitivity when he occasionally took a short cut through the greenhouse.

Just as important as a thorough investigation into the habits of the patient's life is (1) a knowledge of the special characteristics of substances causing contact dermatitis, and (2) acquaintance with the distribution of lesions caused by those substances.

1. SPECIAL CHARACTERISTICS OF SUBSTANCES CAUSING CONTACT DERMATITIS

The substances may be divided into the following groups:

- | | |
|----------------|-------------------|
| (a) clothing, | (d) occupational, |
| (b) cosmetics, | (e) plants, |
| (c) household, | (f) medicines. |

It will not be possible to mention here all substances, but it will be possible to give a guide to their miscellany.

(a) *Clothing*

Dermatitis on the backs of the feet or toes may be caused by shoes, and due to such constituents as resins, tar, plastic or leather.

Nylon stockings may cause a rash on the inner surface of the upper part of the thighs, backs of the knees, and on the feet and toes (Fig. 6). Nylon hair-nets may cause a rash on the back of the neck. In each case, the basic cause is a dye.

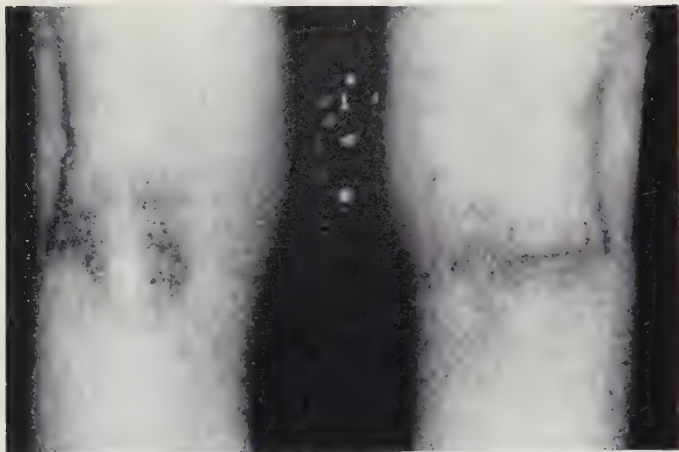


FIG. 6. Nylon dermatitis. Characteristic sites. The soles were also affected (Dr Harold Wilson).

Nickel is one of the commonest causes of contact dermatitis, and is part of chromium-plated suspenders, zips, watch straps and buckles. Nickel is a very strong sensitizer and recovery often takes several years, interspersed with remissions and relapses. Although



FIG. 7. Napkin dermatitis in a subacute condition, showing generalized spread due to secondary eczematization. There was no evidence of moniliasis.

the areas in contact with nickel produce a dermatitis, it has an eccentric habit of secondarily attacking areas, such as the eyelids, and flexor surfaces of the elbows, which have never been in contact with it. The patient may therefore focus an unknowing doctor's attention on these areas, without informing him of a small patch, for example, under a suspender clip.

Napkin dermatitis is one of the commonest skin diseases of infants. It is an erythematous and papulovesicular dermatitis, which occurs on the genitals, buttocks, thighs and lower abdomen, and in severe cases may spread to involve the chest and face, and legs and heels (Fig. 7).

It is usually due to prolonged wearing of wet napkins, and less often to ammonia being formed in the wet napkin, as the result of the growth of a saprophytic bacillus splitting the urinary urea into ammonia; sometimes the cause may be moniliasis.

Disposable napkins should be used, the skin cleansed only with olive oil, and steroid creams or zinc and castor oil applied. The baby should be kept lying on its abdomen as much as possible.

(b) *Cosmetics*

There is no article amongst these substances which cannot produce a dermatitis. The risk of them so doing, however, is extremely slight.

Lipstick is the commonest cause of dermatitis due to cosmetics. It is due to the eosin dye. Nail varnish is the next worse offender. The rash is due to contact from nails recently varnished. The habit of attempting to arrest a stocking ladder by touching it with varnish may, in occasional cases, be the beginning of this type of dermatitis. Both non-allergic lipsticks and nail varnishes can now be bought.

Hair dyes may produce a severe dermatitis, the face being more affected than the scalp, accompanied by oedema of the eyelids, and other areas may be secondarily attacked. Shampoos and hair lacquers can also cause dermatitis, as can perfumes, powders, deodorants, and antiperspirants.

(c) *Household*

Detergents are the commonest cause of dermatitis. They contain soap and synthetic detergents. When their cleaning action is considered it is not surprising to find that they can directly penetrate

the horny layer, and defat the skin. The rash is usually most persistent, and may continue for a long time in spite of treatment. Very few patients can avoid doing household washing, the slightest amount of which is liable to cause the condition to bloom again. For many there is no economic alternative.

Other substances are less likely to cause dermatitis, but such things as polishes and the wearing of rubber gloves may do so.

(d) *Occupational*

The number of causes under this heading is legion. The commonest amongst them are petroleum products, oils, solvents, paint constituents and cement (Fig. 8).

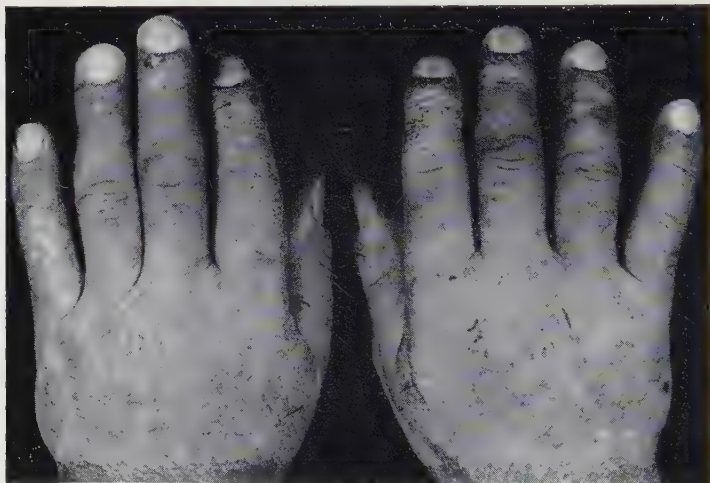


FIG. 8. (a) Cement dermatitis. Scaling, fissuring and crusting (Institute of Dermatology, University of London).

Less commonly, substances such as synthetic rubber, and natural rubber to which chemicals have been added, and natural or synthetic resins which are used in varnish and adhesives, may be the cause. Synthetic resins are more widely known by the name plastics.



FIG. 8. (b) Cement dermatitis. Marked fissuring and scaling.

(e) *Plants*

The commonest plants responsible for dermatitis are primula, chrysanthemums, celery (Fig. 9), tulips and clematis. Daffodils and narcissi are the cause of many cases in the Scilly Isles, amongst pickers and packers.

(f) *Medicines*

The following drugs when applied to the skin are often the cause of dermatitis: sulphonamides, penicillin, streptomycin, antihistamines, the procaine series of local anaesthetics neomycin, framycetin and dequalinium chloride.

In some cases, however, contact with them is unavoidable. For example, nurses and doctors who are obliged to give injections are liable to have small quantities constantly falling on the skin. As the disease establishes itself, it may be impossible for the person to enter a room, a ward, or a theatre where particles of these drugs are in the atmosphere without developing an extremely itchy skin.



FIG. 9. Contact dermatitis. Bullae and vesicles following contact with celery leaves and exposure to sunlight. Also called phyto-photo-dermatitis. It may be caused by other plants, such as parsnips, or various weeds. Patient is a boy wearing short trousers.

2. DISTRIBUTION OF LESIONS CAUSED BY OFFENDING SUBSTANCES

The *head, neck, and face* may be affected by cosmetics, and secondarily in the case of nickel dermatitis. Plants such as primula commonly produce lesions on the face.

The *hands* are the most commonly involved, so that the number of substances capable of causing dermatitis is enormous. Soap, detergents, petrol, oil and cement are some of the common causes.

The *arms* are most often affected by dusts and plants.

The *axillae* may be affected by cosmetics and clothing.

The *trunk* is usually involved by sensitization to such articles as metal clips, or elastic in underwear.

The *feet* may present dermatitis caused by a constituent of shoes or socks.

These lists are not comprehensive, but should serve as a guide to one of the approaches in the diagnosis of the disease.

Patch Tests

When preliminary investigations prove unsatisfactory, these tests must be considered. A patch test is based on the assumption that if a substance causes dermatitis it should produce an inflammatory reaction when applied to an area of unaffected skin.

The method of performing the test is to apply some of the suspected substance to the skin, and cover it with cellophane fixed in position with sticking plaster or 'scotch' tape (Fig. 10). Dilutions of the substance are made for the test.

The test must be carried out during a quiescent phase of the dermatitis; otherwise a very severe reaction may ensue. The patches should be placed in the middle of the back, as pigmentation may result and remain for months in some positive cases.

The patches are left in place for 48 hours, unless itching occurs before this. If negative the site of the patch should be re-examined two days later. Some substances give positive patch tests only if the horny layer has been stripped from the area. This applies notably in neomycin and antihistamine sensitivity.

A negative patch test does not rule out the suspected cause, and positive tests must be judged with regard to other aspects of the history.

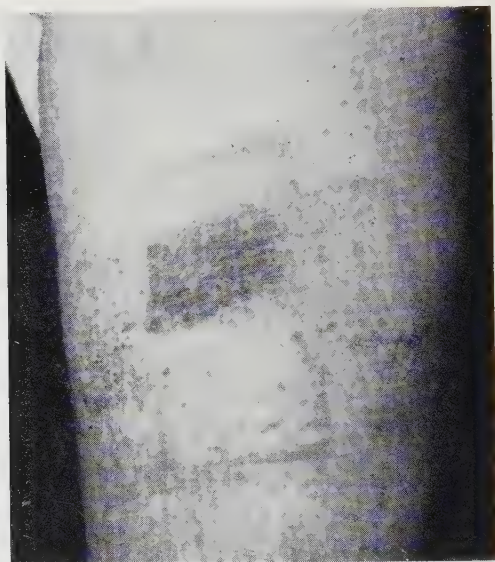


FIG. 10. Patch test showing a positive result, accompanied by a mild surrounding reaction to scotch tape.

TREATMENT

Prophylactic

In industry, workers should be screened before being given jobs where dermatitis is a well-known risk. This is of course very difficult, especially in small factories where medical facilities are slight or non-existent. Even in large building firms, for example, this is not done on account of the endless supply of labourers, so that cement dermatitis is not uncommon.

Working conditions must be good; protective clothing, such as gloves, and the existence of dust-extractors and efficient ventilators are examples of what may be done to reduce the chances of dermatitis.



PLATE I. Psoriasis (Dr G. A. Hodgson).



PLATE 2a. Psoriasis (Dr G. A. Hodgson).



PLATE 2b. Psoriasis.



PLATE 3. Psoriasis: palmar lesions (Dr G. A. Hodgson).

In non-industrial cases, the patient must obviously avoid all contact with the offending substance. Again, it is more easily said than done, as in the case of housewife's dermatitis from soap and detergents.

Curative

The patient must be removed from the source of the dermatitis, otherwise no treatment will cure it, and as a rule avoidance of the cause must be everlasting.

External measures for treating acute cases should consist only of absolutely bland applications, such as saline, Burow's solution, or potassium permanganate 1/8,000 solution. Compresses, with old sheets or hand towels, for 5–10 minutes three times a day, followed by calamine lotion, are effective.

Steroid preparations are not very helpful in the very acute phase.

When the condition becomes less acute, it should be treated as for atopic dermatitis (p. 43).

Internal measures are rarely necessary, and only in very severe cases should steroids be considered. Their use often produces a dramatic result, but it must be remembered that they will not cure the disease if contact is still being made with the offending substance.

Desensitization measures in contact dermatitis have always proved to be very difficult and disappointing.

CHAPTER 5

Erythemato-Squamous Eruptions

The lesions of the following common dermatoses are characterized by redness and scaling. Lupus erythematosus and both tinea and syphilitic infections could be included under the heading of erythemato-squamous eruptions, but will be found elsewhere.

Psoriasis

This is a chronic and occasionally acute inflammatory disease, characterized by papules, or plaques of varying size (Figs. 11, 12; Plates 1, 2). The lesions are reddish and covered with dull silvery scales.

It is found in people who are usually in good health, and is one of the commonest of all skin diseases.

CAUSE

Unknown.

Sex

It occurs equally in both sexes.

Age

Any age may be affected, and it may occur in infants a few months old. The commonest age is between 10 and 30.

Climate

It is commoner in northern climates and winter.

Heredity

The chance of contracting psoriasis is much greater if one or both parents or other ancestors have the disease, than if there is no relevant hereditary history. The familial incidence of all cases is about 30 per cent.



FIG. 11. Characteristic lesions on the front of the leg (Institute of Dermatology, University of London).

Predisposing causes

- (1) Trauma, such as a laceration of the skin. (2) Acute infections, such as tonsillitis: this sort of history is not uncommon in children. (3) Psychological upsets.

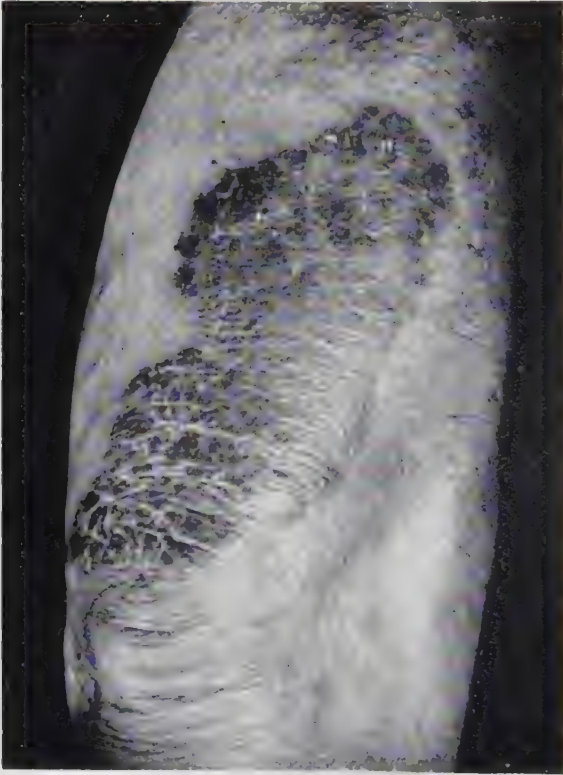


FIG. 12. Psoriasis.

PATHOLOGY

Parakeratosis is present, accounting for the clinically scaly appearance. Thinning of the upper areas of the papillae of the malpighian

layer, with consequent lengthening of the rete pegs, results in blood vessels of the dermis lying closely adjacent to the parakeratotic area and altogether very much closer to the surface than in normal skin. When scales are forcibly removed, pin-point bleeding results because of the proximity of the blood vessels to the surface.

CLINICAL FEATURES

There are usually no symptoms, apart from slight itching in rare cases. The onset is gradual, occasionally explosive. The lesions are papular,

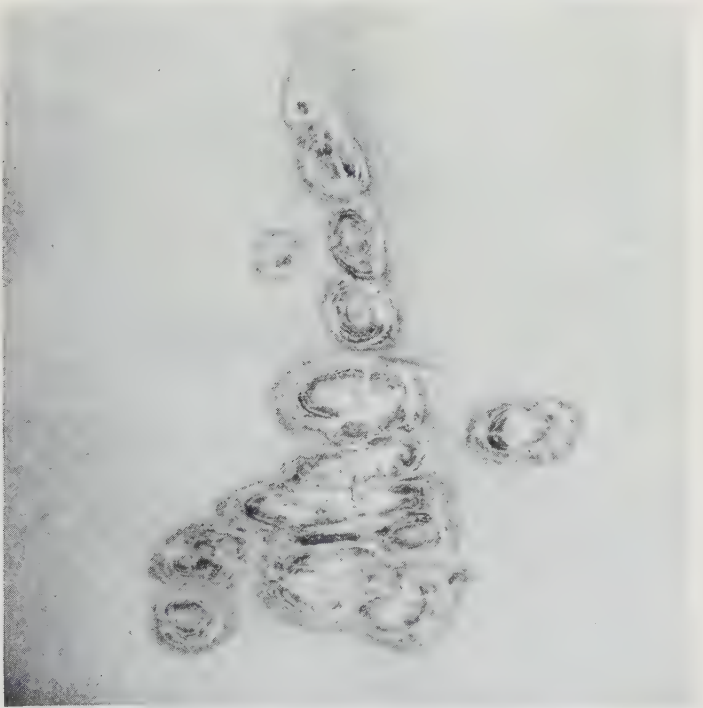


FIG. 13. Psoriasis. Heaped-up inveterate scales in the lumbo-sacral area. No other lesions were present.

dry and scaly. Their size is at first very small, even pin-point, and increases to various magnitudes; in some cases, a sheet of scales may cover half the trunk. Their shape is usually roughly circular, but many bizarre variations occur. The edges are always well defined. The surface is scaly, but stops short near the edge, where it is red. In early lesions, the scale is thin and as time passes the layer of scales thickens (Fig. 13). When scales are removed the underlying surface is



FIG. 14. Psoriasis. Characteristic situation (Institute of Dermatology, University of London).

shiny, red, and very smooth. Scraping of this surface results in the development of pin-point haemorrhages (see Pathology). The colour of the lesions is red, and as they resolve, the colour is less vivid.

Psoriasis characteristically attacks extensor surfaces, such as knees and elbows, but any area may be involved (Fig. 14, Plate 3), and after the extensor surfaces, it is found on scalp, sacral area, chest, face (Plate 4), abdomen, and genitalia. Scalp lesions are thick and round and better felt than seen. Under the palpating finger they are firm and the surface does not crumble as it would were it a case of a crusted lesion.

The nail surfaces are attacked in about 30 per cent of cases and show pitting, like a thimble's (Fig. 15), and may be thickened at the



FIG. 15. Psoriasis. Thimble pitting (Institute of Dermatology, University of London).

free edge (Fig. 16). At the edge, a dirty brownish yellow staining may appear, elliptical or crescentic in shape, sometimes later involving the entire nail (Plate 5). Psoriasis may attack one nail or many, and in rare cases, nail involvement may be the only sign of the disease. Thimble-pitting is seen in other conditions, notably eczema.

Mucous membranes of the lips are very rarely involved.

The number of lesions varies from one or two to hundreds.



FIG. 16. Psoriasis. Thickened and deformed nails, with skin involvement (Institute of Dermatology, University of London).

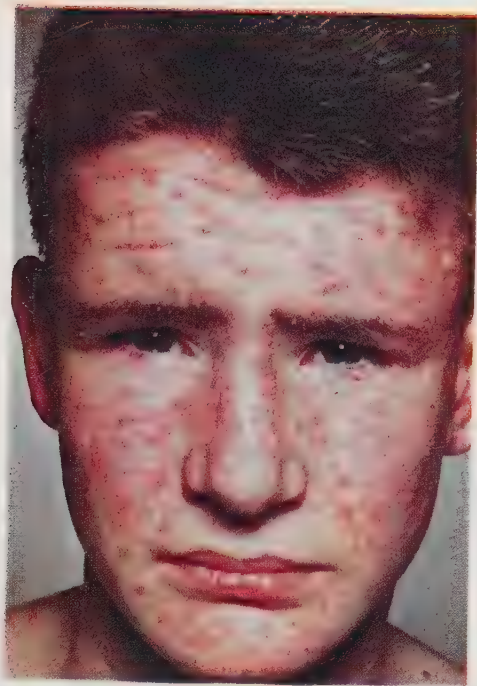


PLATE 4. Psoriasis:
facial lesions
(Dr G. A. Hodgson).



PLATE 5. Psoriasis
(Dr G. A. Hodgson).

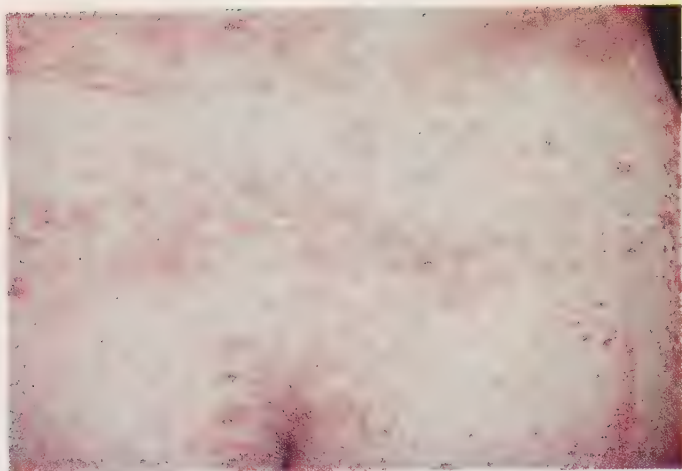


PLATE 6. Pityriasis rosea, showing the rose colour of this condition, in a case in which lesions were generally smaller than usual (Dr G. A. Hodgson).



PLATE 7. Lichen planus. Violaceous, shiny, slightly scaly, penile lesions (Skin Department, London Hospital).

Special features

Koebner's phenomenon

This term is applied to lesions of psoriasis which develop at the site of an injury, for example, a deep scratch being followed by psoriasis in the scratched area. It can only be produced when new lesions are appearing or old ones spreading. This phenomenon may also be seen in lichen planus, warts, and very occasionally in eczema.

COURSE

As the lesions start to clear, normal skin first appears in the centre, and in this state, to the untutored eye, might be considered to be ring-worm. Lastly, the residual margin clears. It is impossible to predict the course of the nail affection, which often resists all forms of treatment.

Variations of psoriasis

1. Arthropathic: in this type, severe polyarticular arthritis involving both large and small joints and resembling rheumatoid or osteoarthritis is found, in conjunction with severe psoriasis.

2. Pustular: sterile firm pustules are found on the palms and soles with or without the usual psoriatic lesion elsewhere.

3. Erythrodermic or exfoliative: the most acute form of psoriasis, in which the entire skin is red and covered with fine scales, except for a few diffusely-spread typical lesions.

Diagnostic aids

Biopsy.

DIAGNOSIS

This is made by the character of the lesion, and the involvement of the extensor surfaces.

For differential diagnosis see Table 1, p. 78.

TREATMENT

In no other condition, except eczema, is a more confident and optimistic approach required on the part of the doctor than in the management of this disease. Otherwise cure will be slow, and relapses common.

The patient's *general health* must be considered, *foci of infection* eradicated, and any *anaemia* corrected.

External

For chronic cases tar preparations are best for body lesions. The following regime is simple and not messy. (1) A daily bath (usually 25 gallons of water) with 6 tablespoonfuls of liquor picis carbonis (tar) added to it. The patient lies in it for 10 minutes, and whilst immersed gently rubs off scales. (2) Later the same day exposure to ultra-violet rays (u.v.r.). (3) After u.v.r., Liq. Picis Carb. paint is applied to the lesions. Tar or dithranol ointment may be used in place of paint.

When this special regime is not used, it is worth applying a steroid cream, such as synalar (fluocinolone acetonide), and to enhance its effect, enveloping the affected area with a polythene covering (see p. 26).

The scalp lesions require tar ointments combined with shampoos. Nail lesions usually resist treatment. For acute cases, nothing but bland creams such as calamine or eucerin should be used.

Internal

Steroids by mouth are useful in arthropathic cases, and in carefully selected cases of widespread chronic and acute psoriasis. The use of folic acid antagonists, such as Methotrexate, may be beneficial, in cases resistant to other treatments. But one should be reluctant to use such potentially dangerous drugs in the treatment of psoriasis. In obstinate single lesions, intralesional steroids may be effective.

PROGNOSIS

A permanent cure is impossible. Where there is a marked familial tendency, and/or a ready psoriatic response to emotional upsets, relapses are more likely. In general, the greater the faith of the patient in the doctor's handling of the disease, the longer are the periods of freedom from it.

Pityriasis Rosea

This is a mild inflammatory disease characterized by macules, and maculo-papular lesions, which are slightly scaly and formed mostly on the trunk (Fig. 17).

CAUSE

Unknown.



FIG. 17. Pityriasis rosea showing the common involvement of trunk, with scattered lesions on the arms and thighs (Department of Dermatology, Addenbrooke's Hospital).

Age

Appears at any age, but is commoner in young adults.

Season

Any time of year, but usually seen in spring and autumn.

CLINICAL FEATURES

Symptoms

Usually no symptoms, but sometimes headache and malaise. Onset is sudden, with appearance of a solitary macular lesion. This first lesion is known as the 'herald patch' and there is an interval of a week or ten days before other lesions appear, while the 'herald patch' persists alone. If it appears on the back, it may not be noticed by the patient. Very occasionally it fails to appear.

Lesions are macular, some are slightly raised, becoming maculo-papular (Fig. 18). The size varies from pin-head to penny size or larger. Their shape tends to be oval, with the long axis running along the lines of cleavage of the skin, so that a streamlining effect is noticed. The edges are quite well defined. The surface is always scaly. In early lesions, the scale is restricted to the centre, the actual edges being free of scale, and later the scale breaks, leaving a gap in the centre. In older lesions, the scale is markedly at the edge, scalloped,

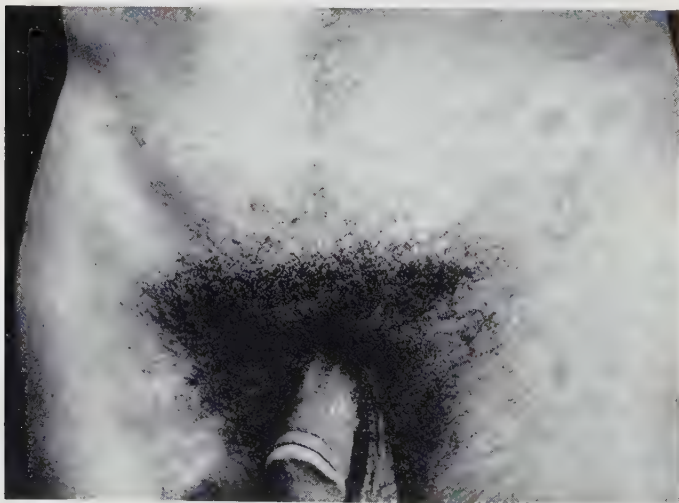


FIG. 18. Pityriasis rosea. Superficial, maculo-papular, slightly scaly lesions (Institute of Dermatology, University of London).

and forming a delicate collarette (Fig. 19). The colour is rose or red (Plate 6). The sites are characteristically the trunk and upper half of the arms and legs. Only occasionally are the lower halves of the limbs, the palms, face, and scalp involved, and then most commonly in children. The number of lesions varies from several to hundreds.



FIG. 19. Pityriasis rosea. Close-up of lesions showing typical collarette of scales (Department of Dermatology, Addenbrooke's Hospital).

COURSE

A week or so after the 'herald patch', many lesions appear and multiply for two or three weeks. Resolution then occurs within six to ten weeks of the onset.

DIAGNOSIS

By the character and distribution of the lesions. For differential diagnosis see p. 78.

TREATMENT

Calamine lotion may be required to suppress itching in a few cases. The patient should be told that the disease is not infectious and that the prognosis is good.



FIG. 20. Seborrheic dermatitis. Scaling also involved the scalp (Institute of Dermatology, University of London).

PROGNOSIS

This is excellent. Relapses and second attacks are very uncommon.

Seborrhoeic Dermatitis

This is an acute, subacute, or chronic inflammatory scaly disease of hairy areas, well endowed with sebaceous glands (Fig. 20). Seborrhoeic dermatitis of the scalp is basically an increase in the normal amount of scaling of the epidermis, which is known as *dandruff* or *pityriasis capitis* (pityron-bran), superimposed by inflammation. Seborrhoea may or may not be present. Other areas attacked by seborrhoeic dermatitis are the axillae, sub-mammary folds, umbilicus, groins and natal cleft. The cause is unknown.

CLINICAL FEATURES

The onset is gradual. The lesions are macular or papular, pin-head to sixpenny size, rounded, and greyish or dirty yellow in colour (Fig. 21). The surface consists of dry scales, some of which can be rubbed off. When the external ear is involved it becomes crusted and may show fissuring at the back. Blepharitis may be present. When the other areas mentioned above are affected, the lesions are similar to those on the scalp.

The condition may be complicated by *eczematization*, when all the lesions suddenly produce a sero-purulent exudate.

DIAGNOSIS

This is made by the character of the lesions involving seborrhoeic areas. Differential diagnosis is described on page 78.

TREATMENT

Scalp. Shampoos should be given daily until the head is free from crusts; and then twice weekly. Sebbix and Genisol shampoos, and spirit soap shampoos are useful. For severe cases, Alphosyl cream (a coal tar extract) or ung. picis co. should be applied daily, for less severe ones, salicylic acid lotion B.P.C. When eczematization has developed, evidenced by extensive oozing and crusting, combined applications of hydrocortisone and terramycin should be made two or three times a day, until the intense inflammation has subsided, before applying creams or lotions.

Face and Ears. A hydrocortisone lotion with or without an antibiotic is usually effective. If it fails, recourse should be made to Salicylic acid ointment B.P. with 2 per cent sulph. ppt.

Body. Treatment as for face.



FIG. 21. Seborrhoeic dermatitis. Polycyclic lesions, suggesting tinea (Institute of Dermatology, University of London).



PLATE 8. Lichen planus. Violaceous, scaly, shiny lesions on the forearm (Dr G. A. Hodgson).



PLATE 9. Lichen planus.



PLATE 10. Lichen planus, showing white patches on the buccal mucosa, and more discrete lesions on the tongue (Dr G. A. Hodgson).



PLATE 11. Lichen planus, showing lesions of the hypertrophic variety on the front of the leg (Dr G. A. Hodgson).

PROGNOSIS

Generally good, although recurrences are not uncommon, especially as regards scalp lesions. For the scalp, therefore, it is advisable to apply the lotion advised above periodically, and to shampoo the scalp regularly. The immediate outlook in acute cases is more favourable when over-zealous treatment is avoided.

Lichen Planus

This is an acute or chronic inflammatory disease, characterized by papules which are flat-topped, polygonal, shiny, violaceous and slightly scaly, found chiefly on the flexor surfaces (Plates 7, 8). Itching is normal.

CAUSE

This is unknown.

Sex

Women are more affected than men.

Age

Adults are most commonly affected, although no age after 10 is exempt.

Predisposing factors: 1. Psychological upsets often precede an attack. 2. Trauma. A scratch or more serious injury sometimes produces the first lesion.

PATHOLOGY

Hyperkeratosis, increase of the granular layer, irregular acanthosis often showing a saw-tooth appearance, and destruction of the basal layer by a band-like infiltrate, which hugs the epidermis. Oral lesions show a similar picture.

CLINICAL FEATURES

Itching may be slight or severe, and is usually intermittent. The onset is sudden or gradual. Lesions are papular, dry and slightly scaly. Their shape on close examination is polymorphic and polygonal (Fig. 22, Plate 9). Their size is at first pin-point, and increases up to pea-size. Later, neighbouring lesions may join, forming large patches of varying shapes. The surface of many small lesions is dimpled, and all lesions are scaly; the scale in some may be very thin and



FIG 22. Lichen planus. Polygonal scaly papules on the wrist and palm, with one annular lesion (Institute of Dermatology, University of London).

adherent, making it difficult to observe, but gentle friction will elicit it. Shininess of the surface is a diagnostic feature, but the lesion may have to be examined from different aspects in good light to reveal this sign.

The colour is essentially red at first, altering in several weeks to violaceous and as the disease clears, to dull brownish. The density of these colours is often interrupted by fine milky-looking streaks and dots, especially in the early phase. They are known as Wickham's striae, and are useful aids to diagnosis.

Sites most commonly attacked are the front of the wrists, the ankles, medial side of the thighs and waist. Lesions are seldom found on the scalp, face, palms or soles. Mucous membranes are affected in 25 per cent of all cases. The lesions on the inner sides of the cheeks are tiny milky-white papules, or are arranged in a network pattern of thin thread-like streaks. The lips, tongue (Plate 10), and palate may be similarly affected.

The nails are affected in 5–10 per cent of cases. The changes are non-specific and may appear as longitudinal ridging of the surface of varying degrees of severity with brownish discoloration, or shedding of one or several nails. The loss may be permanent, or there may be some regrowth.

Special features

Koebner's phenomenon is common (see psoriasis).

Some other clinical varieties

(1) Lichen planus hypertrophicus. In this type the lesions are much thicker than in the common variety and at first glance seem to be warty. They are usually found exclusively on the fronts of the legs. There is no dimpling and shininess is not obvious. At the edge of these hypertrophic lesions (Plate 11), the typical flat-topped, polygonal, polymorphic papules of lichen planus may be found. Itching is present.

(2) Lichen planus bullosus. In this variety, vesicles and bullae appear during the course of the disease.

Diagnostic aids

Biopsy.

DIAGNOSIS

By character of lesions. For differential diagnosis see page 78.

TREATMENT

Attention should be paid to the general health of the patient, especially with regard to any emotional stresses that exist, but treatment does not seem to have much effect on the course of the disease.

External

Hydrocortisone lotions or sprays, calamine lotion, or a 2 per-cent tar ointment may alleviate itching. Superficial X-rays and grenz rays are useful for the hypertrophic variety, when all else fails.

Internal

Vitamin B₁ 50 mg b.d. is sometimes useful. Steroids are beneficial in acute cases and often in the hypertrophic variety. Intralesional steroid injections into those of the hypertrophic variety are also sometimes effective.

PROGNOSIS

This condition usually lasts six months to two years. Relapses may sometimes occur after apparent cure.

Exfoliative Dermatitis

This condition is characterized by generalized scaling and redness. It is usually a complication of a pre-existing skin disease, or drug intoxication. It may very rarely appear primarily, by itself.

CAUSE

Age

It is most common in middle life.

Sex

More common in males than females, the proportion being about three to one.

Other conditions

Psoriasis. Eczema. Seborrhoeic dermatitis. Reticuloses, such as leukaemia, or Hodgkin's disease. Drug intoxications such as arsenic (administered orally, by injection, or as vaginal pessaries), sulphonamides, penicillin, gold.

CLINICAL FEATURES

A constant feeling of being cold, associated with shivering and fever, is characteristic. Itching is variable, and may be intense.

The onset is gradual. Lesions are macular. Their size is at first coin to palm-size, but soon lesions coalesce to form large patches until there is little or no normal skin to be seen. The surface is covered with small thin scales, or large sheets of scales. Occasionally an uneven yellowish exudate may appear. The colour of the entire skin surface becomes very red. The sites involved may be anywhere on the body. The scalp lesions become matted and crusted. The hair loses its normal lustre, becomes brittle, and falls, so that thinning is noticeable. The nails cease growing normally, are dull in appearance and in severe cases may be shed.

COURSE

This varies, and waxing and waning of lesions is characteristic. Recurrences are common, although treatment by steroids has reduced their frequency.

DIAGNOSIS

This is made by the redness of the skin, the diffuse scaling, and constitutional symptoms. It must be differentiated from psoriasis. The essential diagnosis is the condition underlying the exfoliative dermatitis.

TREATMENT

Rest in bed, with particular attention to the avoidance of bedsores which are most likely to occur in the elderly patient. A high protein diet should be maintained.

External

Oatmeal baths daily, provided the patient is fit enough to endure them. A bland greasy application such as Eucerin or thovaline cream is useful. Drying lotions or sprays should not be used.

Internal

Steroids are particularly beneficial in these cases. Treatment of the underlying condition must be kept in mind.

PROGNOSIS

This depends to some extent on the nature of the primary condition. Recurrences and relapses should be carefully watched for in exfoliative dermatitis.

TABLE 1
Differential diagnosis of the commonest scaly conditions

	<i>Character of scale</i>	<i>Distribution</i>	<i>Itching</i>	<i>Onset</i>
Eczema-Dermatitis	Dry, fragmented covered lesion	Any	Present	Variable
Psoriasis	Dry, silvery when scratched; do not reach periphery of lesion	Extensors	Absent	Gradual
Seborrhoeic Dermatitis	Dry or greasy, greyish or dirty-yellow	Hairy areas and creases	Absent	Insidious
Pityriasis Rosea	Dry, broken in centre of lesion, forms a frill at edge	Trunk	Absent	Sudden
Tinea	Dry, peripheral Some residual scales may remain in centre of lesion	Any	Slight	Sudden or gradual depending on type of fungus present
Lichen planus	Adherent, and thin	Flexors	Present	Sudden or gradual
Lupus erythematosus	Adherent, with plugging of follicles by scales	Face, scalp, ears. Rarely elsewhere	Absent	Gradual

CHAPTER 6

Erythematous Eruptions

The conditions described in this chapter are all mainly characterized by erythema. Erythema is a redness which fades on pressure but which returns rapidly on release.

Toxic erythema

The cause is usually hypersensitivity to such foods as shell-fish, mushrooms, or strawberries; or to things such as penicillin or barbiturates; or to external causes, such as trusses or splints.

The eruptions are generally bilateral and symmetrical, affecting the trunk, face and upper parts of the limbs. They are usually morbilliform, or scarlatiniform. General malaise and slight pyrexia are present and the attack clears up in a week or so.

DIAGNOSIS

This is made from specific fevers. Drug eruptions should also be considered.

TREATMENT

The cause is treated if found. Otherwise simple measures such as a calamine lotion suffice.

Erythema Multiforme

This is an acute inflammatory condition characterized by macules, papules and vesicles, symmetrically distributed. Generally, it should be considered as a toxic eruption.

CAUSE

In many cases this is undiscovered but considered to be possibly viral. In this type it is commoner in spring and autumn, and occurs in children.

In other cases, it is secondary to the following conditions:

(i) Drug intoxication—antibiotics such as sulphonamides and penicillin, quinine, salicylates.

(ii) Systemic diseases, such as glandular or rheumatic fever, pneumonia, nephritis or meningitis.

(iii) Serum sickness, following the injections of antitoxins.

(iv) Pregnancy is sometimes accompanied by it between the 5th and 7th month; the eruption fades early in the post-natal period.



FIG. 23. Erythema multiforme. Bullous lesions.



PLATE 12. Erythema multiforme (Dr Martin Beare).



PLATE 13. Erythema nodosum. Bright-red, smooth lesions, in this case associated with acute follicular tonsilitis.

PLATE 14. Erythema
induratum.



PLATE 15. Rosacea.
Well-developed case
with pustules on
forehead and nose.



PATHOLOGY

The blood vessels are dilated, surrounded by cellular infiltration. When vesicles or bullae are present, they are uni- or multilocular, sub-epidermal. Acantholysis is absent.

CLINICAL FEATURES

In most cases there is a prodromal phase of malaise; in severe cases, sore throat, diarrhoea, and pyrexia. In some cases, no symptoms occur.

The onset is sudden. Lesions are macular and papular. Their size varies from pin-point to pea-size; later coalescence of the lesions may occur. Their shape is round and sometimes like an iris, the centre being darker than the rest of the lesion (Fig. 24). In some the surface may show vesiculation and bullae in severe cases (Fig. 23). The colour is red, but always disappears on pressure.

Backs of the hands and forearms are the sites most commonly attacked (Fig. 24). The sides of the neck, the face and legs, genitalia, and mucous membranes may also be involved. The distribution is usually symmetrical.

COURSE

The disease usually lasts 3–4 weeks.

OTHER VARIETIES

The Stevens-Johnson Syndrome

is also known by the more descriptive term of erythema bullosum malignans. It is similar to erythema multiforme, but the clinical features are far more severe, the most marked ones being high fever, widespread and painful involvement of the mucous membranes and joint pains. This condition may be fatal.

DIAGNOSIS

This is made by the redness of the lesions, the iris lesions when present, the bilateral distribution, and the absence of itching.

Urticaria presents itchy wheals and these are rarely symmetrical. When vesicles or bullae are present, *pemphigus* has to be excluded. The bullae of pemphigus arise from normal skin, those of this condition from red macules. *Dermatitis herpetiformis* is an itchy vesicular or bullous eruption.



FIG. 24. Erythema multiforme. Involvement of the hands, showing some iris-like lesions (Dr E. Waddington).

TREATMENT

If the condition is secondary to another cause, the latter should, of course, be treated. Rest in bed is essential in severe and preferable in moderate cases. There is no specific treatment.

External

Eusol lotion or aluminium acetate 5 per cent lotion may help.

Internal

Broad-spectrum antibiotics such as the tetracyclines may be required to suppress secondary infection. In severe cases, especially the Stevens-Johnson syndrome, steroids by mouth are indicated.

PROGNOSIS

Mild and moderate cases are liable to recur once a year for a few years.

Erythema Nodosum

This is an acute inflammatory condition characterized by painful nodules usually on the fronts of the legs (Plate 13), but occasionally on the outer sides of the forearms. It is *symptomatic* of a bacterial, viral or fungal disease, a drug eruption, or a concomitant condition. Streptococcal conditions are probably the commonest cause.

CAUSE

Age

It occurs commonly between 10 and 30 years.

Sex

Females are more affected than males.

Seasons

It occurs more commonly in spring and autumn.

Associated conditions

Streptococcal, as mentioned above. Rheumatic fever, sarcoidosis, tinea, tuberculosis, syphilis, leprosy, and others.

Drugs

Iodides, bromides, sulphonamides, and penicillin especially.

PATHOLOGY

There is a heavy infiltrate in and around the vessels of the dermis, with a few lymphocytes and plasma cells. The infiltrate is also found in the subcutaneous fat. Streptococci or other bacteria may be seen.

CLINICAL FEATURES

The onset is acute, with malaise, fever, and some joint pains. The lesions are nodular, being painful, red and shiny. They reach their full size, about 2–5 cm in diameter, in 24 hours and last about two weeks, healing without scarring. The lesions appear in crops, which is characteristic.

DIAGNOSIS

This is made by the symmetry and tenderness of the lesions. The condition must be distinguished from erythema induratum (Plate 14), which occurs on the calves of the legs, rarely in crops, and tends to ulcerate.

TREATMENT

The cause must be sought, and treated. Cool aluminum acetate compresses are soothing.

PROGNOSIS

The disease lasts about 3–4 weeks, and rarely recurs.

Urticaria

This is an acute or chronic disorder characterized by wheals and papules (Fig. 25), accompanied by itching and pricking sensations. It is also known as nettle-rash.

CAUSE

This is often very difficult to find, and in 50 per cent of cases remains undiscovered.

Sex

Both sexes are equally affected.

Age

Any age may be affected, but it is commoner in the first two decades.

Allergy

This is the commonest cause of urticaria. Allergy is an acquired, altered sensitivity and specific in character; that is to say, it reacts to only one particular substance, or one that is chemically related. The reaction is immunological, based on antigen-antibody reactions, although there may be no means of demonstrating antibodies. Many different substances may be included under this heading:

- (i) Foods: e.g. shell-fish, strawberries, pork, eggs.
- (ii) Drugs: penicillin, aspirin, enemas, codeine and others.
- (iii) Parasites: fleas, lice, intestinal worms.
- (iv) Physical agents: heat or cold.
- (v) Inhalants: house dust, feathers.



FIG. 25. Urticaria showing papules and wheals (Institute of Dermatology, University of London).

Psychogenic

This group accounts for quite a large proportion of cases of chronic urticaria, anxiety neuroses and emotional conflicts commonly being responsible. Conditions which give rise to one of these two states are common enough to be part of the daily life of a great many people at one time or another, and therefore these factors as a cause of urticaria must be considered with care.

PATHOLOGY

Vascular dilatation occurs in the dermal vessels, with an outpouring of serum and white cells. This collection of fluid compresses the vessels, so that there may be blanching at the centre of the wheal. As the fluid increases, it makes its way through the epidermis to produce a vesicle, or bulla. These phenomena occur as a result of liberation of histamine in the skin.

CLINICAL FEATURES

Itching, burning and pricking sensations are constant. The onset is sudden. The lesions are wheals. Their size varies from a pea to palm size or larger. Their shape also varies and may be very irregular when coalescence of lesions occur. The surface may be smooth, vesicular or bullous, and they are red, although central blanching may occur. The number may be few or many. Any area may be involved, but arms, legs, thighs and waist are the most favoured. The mucous membranes are also commonly attacked, especially the larynx.

Special features

Dermographism is common in urticaria (Fig. 26), and is characterized by the ease with which wheals are provoked by rubbing the skin with a blunt instrument. It can be more easily produced in acute than in chronic urticaria.

Laboratory findings

Eosinophilia is common, but inconstant.

COURSE

Acute urticaria clears up in one or two weeks; chronic urticaria may persist for weeks, months or years.

Other clinical varieties

(i) Bullous, often seen in children. (ii) Giant urticaria, or angio-neurotic oedema, which differs from ordinary urticaria only in the size of the lesions. In this type, the eyelids, lips, and the lobes of the ears are most commonly involved. When the eyelids are affected the patient may find it nearly impossible to see, and when the lips are affected eating may cause great discomfort.

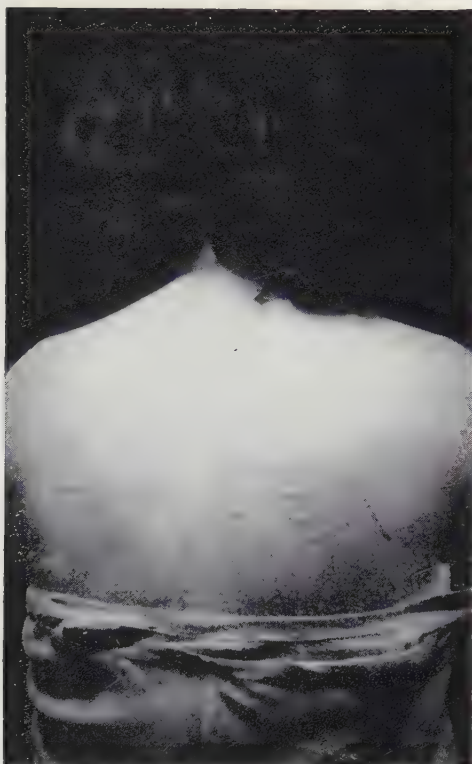


FIG. 26. Urticaria demonstrating dermographism.

DIAGNOSIS

This is made by the presence of wheals. *Scabies* must be excluded, by a search for burrows.

TREATMENT

Prophylactic

If the cause can be discovered, it must be avoided; it is usually food or a drug.

External

Oatmeal baths are soothing. Calamine steroid lotions and sprays are palliative.

Internal

Many anti-histamines are effective and are given in liquid form to children, in tablet form to adults. For adults, chlorpheniramine maleate (Piriton) 4 mg t.d.s., or Triprolidine (Actidil) 2.5 mg t.d.s. may be given. For children these doses are graduated according to age.

Oral steroids may have to be given for severe cases of giant urticaria. In ordinary chronic cases they should only be used after considerable thought. There is no doubt, however, that they are helpful.

PROGNOSIS

This is good in acute urticaria but should be guarded in chronic cases, although this type does eventually clear up.

Rosacea

This is a chronic inflammatory disorder, sometimes with acneiform lesions, and telangiectases involving the central areas of the face.

CAUSE

This is not exactly known, but anything giving rise to persistent reflex flushing of the face provokes its development.

Sex

Far more common in women.

Age

From the age of 30 and in women most noticeably at or about the menopause.

Predisposing Factors

Occupation. Those exposed to a great deal of sunlight or severe winds, such as farmers and sailors, are very prone.

Food. Chronic alcoholism and a spicy diet both aggravate rosacea.

Psychological. In many cases there seems to be a relationship between emotional upsets and the production of rosacea, especially in instances of chronic frustration or depression.

PATHOLOGY

There is dilatation and the formation of new capillaries which account for the redness of the lesions. There is also a dermal lymphocytic infiltrate, which sometimes gives the lesions a rather brownish colour. The sebaceous glands are occasionally hypertrophied, which produces rhinophyma (see below).

CLINICAL FEATURES

The first symptom of the disease is usually bouts of hot flushing of the face, which may be increased by exposure to sun and wind, or by imbibing hot or alcoholic liquids.

The onset is gradual. Lesions are papular. Far more conspicuous is the redness of the face. The chin, cheeks, nose and forehead may be involved, severally or singly. The cheeks and nose are known as the butterfly area of the face (Plate 15). In this acneiform condition there are no blackheads.

Other varieties

Rhinophyma may develop. This gross hypertrophy of the nose is associated with easily visible pitting and marked redness. It is commonest in men, and alcoholics.

Complications

Conjunctivitis, iritis and keratitis may occur. These signs in some cases precede the onset of the facial lesions, but usually follow them.

DIAGNOSIS

This is made by the distribution on the butterfly area of the face and the absence of blackheads. *Acne vulgaris* always presents comedones (blackheads), and involves any area of the face, as well as the chest and back. *Lupus erythematosus* appears as well-defined patches on the face, without acneiform lesions.

TREATMENT

Predisposing factors must be avoided as much as possible. Rest periods should be increased and regularly taken.

External

Sulphur 2 per cent in calamine lotion is nearly always helpful, but if it is not, a steroid lotion may be used.

Internal

Nicotinamide 50 mg t.d.s. p.c. and lactoflavin 3 mg t.d.s. p.c. are often most useful. Broad spectrum antibiotics such as the tetracyclines, when taken for a long period as for acne, may also produce good results. An alkaline mixture such as mist. gent. alk. one tablespoon before meals is indicated if symptoms of dyspepsia are prominent.

Rhinophyma is treated by plastic surgery.

PROGNOSIS

Mild cases usually respond rapidly to treatment, but in others relapses are disappointingly frequent.

CHAPTER 7

Diseases due to Physical or Chemical Substances

Drug Eruptions

There is no categorical method of describing the appearance of a drug eruption, as it may imitate some features of practically any other disease. It is, however, the tendency to produce some but not all features of another disease which should strike one as being bizarre and thus make one consider this diagnosis as a possibility.

Such eruptions are caused by drugs ingested, injected, or otherwise absorbed and should not be confused with those caused by external applications.

CAUSE

Little is known about the exact way in which drugs cause eruptions, but certain theories are accepted.

Allergy seems to produce some reactions, but there are no skin tests to prove it.

Idiosyncrasy in some people leads to the appearance of an eruption and may result from sensitivity to a drug which had previously caused dermatitis through topical application. The dermatitis, a thing of the past, may have been forgotten by the patient. This applies especially to sulphonamide and penicillin ointments, which are never now used by dermatologists.

Any *age* may be affected, females more than males, and *highly-strung individuals* are more prone.

CLINICAL FEATURES

The onset varies with the drug concerned, from sudden (e.g. penicillin) to attenuated (e.g. arsenic). The lesions may be urticarial, erythematous, vesicular, bullous, purpuric or pustular.

Distribution is generalized, except in the fixed drug eruption (see below). The colour is often curiously bright. In the case of some drug eruptions the lesions are characteristic and these will be specifically described below. A great many are non-specific and will be grouped under one heading (see below).

DIAGNOSIS

In some cases, the diagnosis can be made at a glance. Typically, the onset is sudden, the rash widespread, bright and symmetrical and the history shows that the taking of a drug is responsible for it. In other cases the diagnosis must be considered when the eruption is distinctly peculiar and does not fit into any known pattern of disease, is bullous, or present as an exfoliative dermatitis.

Having considered the possibility of a drug eruption, it is important to know which common drugs may be associated with the presenting skin reaction and, conversely, to know the peculiar and unique presentations of a few drugs in common use.

A. ERUPTIONS, AND SOME DRUGS WHICH PRODUCE THEM

1. *Urticaria*. Penicillin, aspirin, iodides.
2. *Morbilloform and scarlatiniform*. Penicillin, sulphonamides, chloramphenicol, streptomycin, barbiturates, anti-histamines.
3. *Vesicular and bullous*. Iodides, bromides, sulphonamides.
4. *Purpura*. Sedormid, carbromal, chloramphenicol. An itchy rash develops, especially on the buttocks and legs.
5. *Light-sensitivity eruptions*. Sulphonamides, phenothiazine tranquillizers. The rash is limited to exposed areas.
6. *Exfoliative dermatitis*. Mercury, gold and all heavy metals.
7. *Fixed drug eruption*. Phenolphthalein (in laxatives), antipyrine, and barbiturates.

The lesion is well circumscribed, dusky red, and usually on the trunk, or upper parts of the limbs. It lasts a few days and leaves a hyperpigmented patch. There may be one or more patches and they always reappear in the same site, following the taking of the drug.

8. *Lichenoid*. Anti-malarials, gold, bismuth, amiphenazole, chlorpromazine.

B. SOME DRUGS, AND THEIR TYPE OF ERUPTION

Arsenic, as liquor arsenicalis (Fowler's solution) given over a long period may give rise to pigmentation, keratotic lesions of the palms and soles, and eventually intra-epidermal carcinomata.

Antibiotics of the broad-spectrum variety, e.g. tetracyclines, may produce ano-genital pruritus.

Barbiturates may cause urticarial, erythematous, purpuric, bullous, and fixed eruptions. Aphthae and stomatitis also occur.

Bromides and iodides may cause an acneiform eruption, but without blackheads, which does not occur in true acne. Infants may suffer by transmission of the drug through the mother's milk.

Chloroquine (Nivaquine) may cause difficulty with focusing, and photophobia; retinopathy after prolonged courses; and permanent and progressive retinal damage, although the changes may be reversible in the early stages.

Chlorpromazine (Largactil) may produce various erythemata, with light sensitivity an added factor, and lichenoid eruptions (Fig. 27).

Penicillin. Urticaria is the commonest eruption which may be chronic and severe. Penicillin may also be carried in the milk of cows treated with it, and then may be conveyed to babies via their mother's breast milk. Penicillin is the commonest of all causes of drug eruptions.

Serum can cause urticaria or erythema, one or two weeks after injection.

Steroids may produce hirsuties, hyperpigmentation and acneiform eruptions.

TREATMENT

Most drug eruptions clear up within a week of stopping the drug. The taking of the smallest amount of the drug will result in a relapse. Plenty of fluids should be given to hasten elimination.

Antihistamines, e.g. triprolidine (Actidil) 2.5 mg or chlorpheniramine maleate (Piriton) 4 mg three times a day is useful in the early phase of penicillin urticaria.

Systemic steroids are required for exfoliative dermatitis.



FIG. 27. Drug eruption: lichenoid lesions due to chloroquine.

Sunburn

This is caused by ultra-violet rays from the sun. First or second degree burns may follow a few hours of exposure, blondes being more sensitive than brunettes.

Cold creams or steroid creams are useful as prophylactic and curative measures. Mexenone cream (Uvistat 2211) is also a useful barrier.

Prickly Heat

Miliaria

This condition is characterized by pin-point to pin-head sized vesicles and papules, with pricking and burning sensations, and is due to inflammation of coil-glands.

It is a feature of tropical life, or in those whose work entails exposure to excessive heat.

TREATMENT

Light clothing should be worn. Heavy meals and alcohol must be avoided. Cool bran or oatmeal baths are useful. Steroid sprays are temporarily beneficial.

PROGNOSIS

Once the tendency to prickly heat is established, it can rarely be abolished.

Chilblains

Pernio

This painful condition is characterized by red or nodular lesions on the fingers, toes, ears or face resulting from hyperactivity of the peripheral vessels in response to cold and is commonest in young women. The lesions are also associated with burning and itching and, in some cases, ulcerate.

They may be mistaken for erythema induratum (p. 117) or erythema nodosum (p. 83).

TREATMENT

Warm clothing is essential; cold must be avoided. Toilet lanoline applied in the morning may act as a partial insulator.

A strong erythema dose of irradiation from an ultra-violet radiation lamp, once a week for three successive weeks, in September and October, may protect the treated areas for the ensuing winter (Ingram).

CHAPTER 8

Psychological Factors in Skin Diseases

Introduction

The awareness of the relationship between the skin and the mind, in health and disease, has been evident in literature for the past 2,000 years. To-day, expressions such as 'becoming hot under the collar', 'rubbing the skin up the wrong way', or 'someone getting under one's skin' are further reflexions of the association and of cutaneous reactions to shame, guilt, anger and anxiety.

Nothing actually happens in our minds without affecting our bodies and the reverse is also true, body and mind referring only to two different aspects of the single indivisible whole. When our body or mind is not precariously poised in a critical incident, the relationship between them is scarcely noticed. But when shame, guilt, or anxiety, for example, produce an interruption of the interaction of the body and mind, physical reactions occur which vary from those recognizable by the man-in-the-street, to those whose significance is often variously calculated by differing physicians. Those readily recognizable are an increased pulse rate, vague precordial pain, indigestion or sweating. The physicians have to assess the relationship of heart, stomach or skin symptoms and signs.

There are some apparently substantial reasons for the skin being affected by emotional disturbances. It is obviously used for expression, as blushing or pallor show. It has more pathways leading to the brain than any other organ, and is therefore closely associated with fundamental emotional deviations. The skin is also used in daily life for the reinforcement of confidence by means of beautification and dress.



PLATE 16. Localised neurodermatitis
Well-defined chronic lesion showing superficial adherent and uneven scaling, with some crusting (Dr Martin Beare).

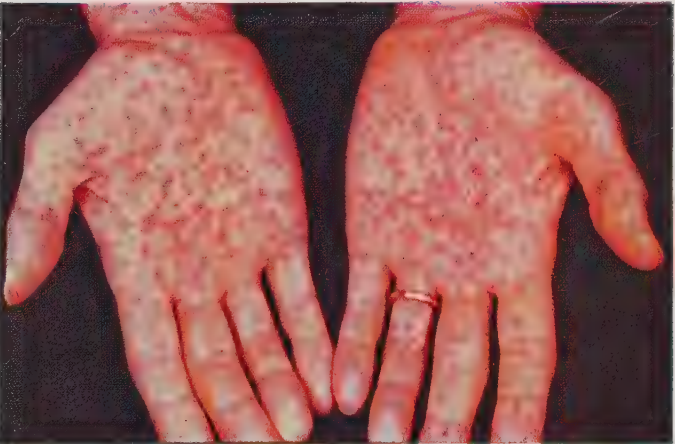


PLATE 17. Secondary syphilide:
papulo-squamous lesions.



PLATE 18. Tinea cruris. Well-defined lesions with a scaly edge (Skin Department, London Hospital).



PLATE 19. Tinea capitis (*T. discoides*) (Dr Martin Beare).

However, very few skin diseases are a DIRECT result of psychological disorders.

They are:

1. Dermatitis artefacta.
2. Trichotillomania.
3. Parasitophobia and other phobias.
4. Neurotic excoriations.

There are, however, many skin diseases which are sometimes triggered off by emotional upsets, but are not directly attributable to them.

Amongst this group are:

1. Allergic conditions.
2. Alopecia areata.
3. Pompholyx.
4. Eczema—dermatitis (hay-fever—asthma syndrome).
5. Hyperidrosis.
6. Localized neurodermatitis.
7. Ano-genital pruritus.
8. Psoriasis.
9. Rosacea.
10. Urticaria.

When investigating a case for emotional factors, and then discovering them, the tendency to lose sight of causal somatic factors is considerable. It is therefore more than ever important first to exclude organic causes. The cases in which organic sources are missed as factors in the cause of skin disease greatly outnumber those in which psychological factors are missed, for the reason mentioned above. In many instances, it is important not to be misled by the patient's conception of the onset of the disease, which seems so felicitously to suit the case. When psychological factors appear to be the pivot around which the cause revolves, the results of psychotherapy are extremely difficult to evaluate, since they are invariably associated with simultaneous topical treatment. It seems therefore that the role of psychotherapy in this group is essentially supportive rather than leading.

Except for the following types of pruritus, the conditions named above have been dealt with elsewhere.

Pruritus

Pruritus is itching, which may be generalized or localized. Pruritus is considered to be due to the stimulation of the subepidermal nerve plexuses, by proteolytic enzymes, released from the epidermis as a result of primary irritation, or allergic sensitization reactions.

Pruritus may occur without any cutaneous lesions, in conditions such as Hodgkin's disease, pregnancy, diabetes mellitus, or senile skin. The greatest number of such cases are due to psychological causes, and it is these factors which should be carefully investigated.

Senile Pruritus

This is probably due to ischaemic and atrophic skin changes. Itching is spasmodic and often mercilessly severe.

TREATMENT

This is usually disappointing. Local treatment such as steroid sprays provide only temporary relief. Sedatives such as phenobarbitone, $1\frac{1}{2}$ grains three times a day, may help. In severe cases it is necessary to give steroids by mouth. Prednisolone, 5 mg three times a day, often produces the greatest respite from itching; after two or three weeks it may be reduced to a minimal dosage of 5 or 2.5 mg daily and eventually stopped altogether.

Ano-Genital Pruritus

Pruritus vulvae, ani, or scroti

Organic causes which must be excluded before psychological factors are considered as follows:

1. Diabetes.
2. Vaginal infections, such as trichomonas vaginalis.
3. Urinary infections.
4. Threadworm infestations, in children.

These can be easily excluded.

Other less usual conditions are:

5. Contact dermatitis from contraceptive sheaths or pessaries.
6. Ringworm.
7. Seborrhoeic dermatitis.

8. Psoriasis.
9. Lice.
10. Menopausal atrophy.

Elimination of these conditions justifies the taking of a history in search of psychological causes.

The cause may be a superficial one easy to deal with, such as a passing anxiety. More often, however, the problems are deep and complicated, involving perhaps a revolt against intercourse, for reasons of incompatible appetites, fear of pregnancy, or the presence of a third party.

In these cases psychotherapy may be invaluable, but must still be accompanied by local treatment, the best of which is in the form of a steroid lotion or cream.

Recurrences are not unusual, and the management depends to a great extent on the confidence of the physician and the patient's acceptance of it.

Localized Neurodermatitis

Lichen simplex chronicus

This is a chronic low-grade inflammatory disorder due to scratching, characterized by circumscribed areas of thickened and very itchy skin.

It is commoner in females than males, and usually starts after the age of 40.

CAUSE

This is unknown.

Psychological factors usually play a part. The condition might be explained as an extension of the scratching of the head or face which often occurs at the end of a day's work when owing to fatigue problems appear to be bigger than they are. Mild nervous tension may be sufficient to trigger off the desire to scratch and produce the disease. Other nervous features commonly exist, such as nail or lip-biting, or chain-smoking of cigarettes.

PATHOLOGY

A thickening of all layers of the epidermis may be seen, a little oedema, and a dermal lymphocytic infiltrate.

CLINICAL FEATURES

Itching is intermittent and sometimes very severe.

The onset is insidious, itching and scratching preceding the appearance of any lesions. Papules are first seen, which become confluent to form circumscribed patches of varying shapes and sizes. The colour is red or reddish brown and the surface may be covered with an uneven layer of superficial scales (Plate 16).

The sites commonly affected are the nape of the neck (especially in women), the outer sides of the forearms, the calves of the legs and the outer side of the ankles; in fact, any area which is easy to scratch. Pruritus of the ano-genital area may also be a symptom of localized neurodermatitis.

Glycosuria is occasionally found, and urine examination should not be omitted.

DIAGNOSIS

The disease must be distinguished from *lichen planus*, the lesions of which are purplish, distinctly shiny, and more discrete.

Contact dermatitis, in which the duration and onset are swifter, can be excluded by taking a careful history.

TREATMENT

If the patient can be made to stop scratching, the lesion will heal and this must be made clear to the patient. In investigating a possible psychological cause, it is important to realize that it may have arisen from an episode in the past, such as nursing a dying parent for a long time, sailing through stormy matrimonial seas, or attempts to dodge the tax-man, and the relationship of cause and effect is more real than apparent. In many instances the cause is co-existent and must be treated.

External measures consist of the application of a steroid lotion or cream, or an occlusive bandage, such as viscopaste, or cortacream, which is maintained *in situ* for a week or two before being reapplied.

X-rays in selected cases are also useful.

PROGNOSIS

Recurrences are common, no matter how successful the therapy appears to have been.

Dermatitis Artefacta

This term describes self-inflicted lesions, varying from redness to ulceration, induced by hysteria or a neurosis. The notable feature about the condition is the absence of complete resemblance to any other disorder, and the curious shapes of the lesions.

CAUSES

Hysteria

This comprises the main group. The patient may deny knowing how the lesions developed, and he may, in fact, not know their cause.

Psychoses

Schizophrenics and paranoiacs may mutilate themselves without any obvious motive.

Malingers

These patients produce the lesions with a motive such as avoiding work which they dislike.

Agents

These may be such substances as carbolic acid, alkalis, cigarettes, matches or sandpaper. They are ingeniously hidden, and it often requires hospitalization before the cause and diagnosis are definitely discovered.

CLINICAL FEATURES

The lesions have an artificial and curiously bizarre appearance possessing angles and edges not associated with the lesions of any other disorder (Fig. 28). Their severity depends on the agent used; severe burns, deep scars, and ragged ulcers may be seen.

TREATMENT

This is usually difficult and depends on the type of patient.

If hysterical, admission to hospital is often necessary. The patient should be carefully watched and the lesions enclosed in occlusive bandages. Lesions developing later on another limb are suggestive of the presumptive diagnosis. The patient should be kept from suspecting the purpose for admission to hospital until proof is



FIG. 28. Dermatitis artefacta, injurious agent unknown (Institute of Dermatology, University of London).

definite. Even when the patient is aware of the doctor's diagnosis, lesions may continue to be produced.

For psychotic patients, psychotherapy is required. For malingerers merely to catch the patient producing the lesion is enough to end the disorder.

Trichotillomania

This disorder is characterized by an uncontrollable desire to pull out one's own hair.

CAUSE

Emotional factors are always present. Mild or severe frustrations and anxieties may exist, or more rarely, a true psychosis may be present.

Children predominate, but adults are also sometimes affected.

CLINICAL FEATURES

Usually in one area of the scalp there is a patch of relative baldness with broken off but otherwise normal hairs.

DIAGNOSIS

This disorder must be distinguished from *alopecia areata* and *ringworm*.

In the first case, exclamation-mark hairs will be found (see p. 204) and in the second case, the fungus.

TREATMENT

Children usually grow out of the habit without any special treatment, but adults may require psychotherapy.

Neurotic Excoriations

A disorder in which scars are produced by the patient picking at the skin.

CAUSE

Emotional factors are always present and more often than not are features of hysteria. Women are more prone to the condition than men, and 20–30 is the commonest age group.

CLINICAL FEATURES

Lesions occur on any site accessible to the fingers.

TREATMENT

Suggestion or psychotherapy are usually partially or wholly effective.

CHAPTER 9

Bacterial Diseases

Introduction

Bacterial infections of the skin are recognized clinically by the presence of crusted lesions, the crusts being the result of dried exudate. The infection may be a primary cause of the skin disease, or it may be superimposed on another skin disease.

Coagulase positive staphylococci are the commonest cause of bacterial infections of the skin. The staphylococcus aureus is present in the nostrils of nearly 50 per cent of the population and in the body creases of at least 10 per cent. These areas are therefore a common source of infection.

Other well-known organisms are capable of producing bacterial skin infections, such as streptococci, coliform bacilli, pseudomonas and proteus, and more rarely, the Klebs-Loeffler bacillus.

The treatment of bacterial infections has of course been revolutionized by the use of antibiotics. The treatment of skin diseases by them differs from other diseases in that sulphonamide, penicillin or chloramphenicol creams or ointments must *not* be prescribed as these antibiotics, when used locally, are liable to initiate a sensitizing dermatitis which will transpire to be far more troublesome to deal with than the original disease. Other antibiotics such as the tetracyclines, e.g. achromycin, neomycin or framycetin, are just as effective and simple to use in creams and ointments.

Impetigo

This is a contagious, superficially inflammatory disease, characterized by thin-walled vesicles and bullae, which rupture and form honey-coloured crusts (Fig. 29). They are caused by staphylococci

or streptococci and occur predominantly in children. It is a more contagious disease in children than in adults.



FIG. 29. Impetigo contagiosa. Crusted lesions on the face, secondary to a septic focus in the nose.

PREDISPOSING CAUSES

are infected nostrils or ears, dirty finger-nails or towels, or infection from an existing condition such as pediculosis capitis, scabies or eczematous conditions.

PATHOLOGY

The vesicle or bulla arises just below the horny layer and contains polymorphs, lymphocytes and fibrin.

CLINICAL FEATURES

Itching is quite common and the resulting scratching leads to inoculation of the disease elsewhere.

The size of the lesion varies from a pea to a coin. The shape of the lesion is roughly circular, but gyrate lesions also occur. Sites most commonly affected are the face and ears.

COURSE

Untreated the disease tends to clear up spontaneously in a month or so.

DIAGNOSIS

When lesions are circinate, ringworm may be considered, but the lesions are never so crusted or red and fungi are found in scrapings.

TREATMENT

An antibiotic ointment, to which the organisms have proved to be sensitive, applied twice daily will clear the disease within a week. Possible sources of infection, such as the nose and ears, should be examined and treated similarly when necessary.

Boil*Furuncle*

A boil is an acute painful infection of a hair follicle caused by *staphylococcus pyogenes*. A *carbuncle* is a conglomerated mass of boils.

PREDISPOSING CAUSES

are eczema, scabies, pediculosis, and seborrhoea which, being itchy conditions, result in the inoculation of staphylococci by scratching. Diabetes mellitus, alcoholism, anaemia and lowered states of health from, for example, overwork, also predispose to boils.

Latent foci in the nose or ears should be looked for.

CLINICAL FEATURES

The onset is quite sudden. The skin becomes red round a follicle; tenderness, heat and oedema follow, and the centre becomes yellow

with pus. It soon discharges and a yellowish-green core usually follows. Scars are a common remainder. Sites commonly affected are the back of the neck, the axillae, the buttocks and the thighs.

TREATMENT

Penicillin may be employed in doses of 250 mg four times a day in tablet form, for 4 or 5 days. Should this fail, as it occasionally does, a broad spectrum antibiotic such as a tetracycline (Aureomycin) or an oxytetracycline (Terramycin) should be used in the same dosage. Local applications are not useful, but the painful boil should be protected from trauma by a dressing.

Recurrent boils

are often a common feature in those subject to boils. The inside of the nose and ears should be treated with neomycin or terramycin ointment twice daily, and also the perineal area, if bacteria have been discovered there. The entire skin should be rubbed over with hexachlorophene (pHisoHex) in a daily bath, and when dry the skin should be dusted with chlorhexidine (Hibitane) powder, thus constructing an anti-staphylococcal barrier.

Any of the predisposing factors must of course be excluded.

Folliculitis Barbae

This is a chronic staphylococcal infection of the hair follicles of the beard and moustache areas. It is also known as barber's rash. Although the title emphasizes involvement of the beard, other hairy areas such as the thighs may also become affected. On the face, the condition is usually secondary to a nasal infection.

CLINICAL FEATURES

The onset is insidious. The lesions are follicular, each pustule being pierced by a hair and once established, the spread to other follicles is rapid. Blepharitis is a common accompaniment.

TREATMENT

of this apparently straightforward condition is often difficult.

Eradicate focal sepsis, such as bad teeth or infected tonsils.

External

Apply the antibiotic to which the organisms are sensitive, e.g. tetracycline (Achromycin) or oxytetracycline (Terramycin) ointment for at least a month.

Internal

The same drugs are used, e.g. Achromycin or Terramycin 250 mg t.d.s. for 1 week, and then b.d.s. for 1 week.

PROGNOSIS

The avoidance of recurrences, which are apt to occur, depends on meticulous hygiene of shaving and other toilet equipment, such as towels, and the daily application of hexachlorophene (pHisoHex) lotion, after shaving.

Syphilis

Lues

This is an infectious and contagious disease, caused by *Treponema pallidum* (*Spirochaeta pallida*), which is capable of invading every organ of the body and producing such protean manifestations that it is likely to imitate many other diseases in many ways. W.H.O. however reported increases again in 70 of 105 countries who reported in 1965.

Since the introduction of antibiotics there has been a great reduction in the number of cases all over the world, and this being so, diagnosis of syphilis of the skin often depends on consciously remembering its existence.

The synonym lues, and its adjective luetic, are useful words to use in front of patients when discussing the possibility of a diagnosis of syphilis, and the patient should not be informed until the diagnosis is an absolute certainty, for the consequences to the patient are very great.

CAUSE

The organism *Treponema pallidum* is a delicate threadlike spiral parasite, with pointed ends, the number of spirals varying between 6 and 15. It can bend on itself without losing its spiral shape, open and close like a concertina, and act like a screw in its movements.

Transmission

in acquired syphilis may be by direct infection through sexual intercourse (in 95 per cent of cases), less commonly by kissing, or much less commonly through contaminated drinking vessels, or the communal use of such things as tattooing needles, or wind instruments.

Congenital syphilis is transmitted by an infected mother through transplacental passage of the organism into the blood stream of the foetus. The father cannot transmit congenital syphilis.

PATHOLOGY

The essential lesion in all phases of syphilis is the same, being strikingly characterized by peri-vascular round cell infiltration, mainly lymphocytic, and to a lesser extent by plasma, and mononuclear cells. Granulomatous and giant-cell formation occurs in later stages, and all these changes give rise to the sensation of induration derived from palpating (with a protected finger) a syphilitic lesion.

The primary chancre and the lesions of secondary syphilis contain many spirochaetes, but in the tertiary or gummatous lesions they are sparse.

CLINICAL FEATURES

There are 3 stages which are quite distinct in the time they occur and the features they present.

Primary stage occurs between 10 and 20 days after infection (the limits are 9–90 days). The primary lesion, or chancre, is a firm roundish, small inflammatory lesion, which oozes with a stringy non-purulent exudate. The site most commonly affected is the coronal sulcus, but any area of the penis may be involved, including the interior of the urethra, and the ano-rectal area in homosexuals. In women, the labium majus or minus are the commonest areas, but no area is immune including the cervix. In a small percentage of cases, the lips or other facial areas may be the site of a chancre. The site of the genital chancre in women often results in it being overlooked, so that secondary syphilis is the first intimation they have of the disease.

Regional adenitis is common, and malaise is often present.

COURSE

Untreated, a chancre will resolve, and the secondary stage be the first sign of the disease.

INVESTIGATIONS

Treponema pallidum is found by microscopical dark-ground examination of exudate from a lesion, or from fluid aspirated from an enlarged regional lymph node.

The Wassermann reaction (W.R.) and Price precipitation reaction (P.P.R.) are usually positive 4–6 weeks after infection, and invariably during the second stage. Also technical errors may occur and positive results should always be confirmed by repetition. It should be noted that the W.R. is often positive in other conditions, such as yaws, leprosy, glandular fever and malaria, and does not therefore invariably indicate syphilis.

DIAGNOSIS

This is made by the investigations described, and the clinical signs.

Scabies of the penis is associated with itching and may be found in burrows when present. *Herpes genitalis* is a very superficial lesion. *Chancroid* lesions are painful and not indurated, and uncommon in this country. An *epithelioma* has a rolled edge, and its evolution is extremely slow.

A chancre of the nipple must be differentiated from *Paget's disease* of the nipple, a digital one from *paronychia*, while *haemorrhoids* or *fissures* may superficially mimic an anal chancre.

SECONDARY STAGE

This occurs between 6 weeks and 2 years after the appearance of the chancre, and lasts about 2 years. About 75 per cent of patients show skin lesions, 50 per cent have generalized lymphadenopathy, a third have oral and tonsillar lesions, and a very small number have involvement of the nervous system, bones, eyes or abdominal viscera.

CLINICAL FEATURES

These are usually vague, and include headaches, nausea, vomiting, anorexia, bone, muscle and joint pains, laryngitis, tonsillitis and fever, usually 100°–101°F (38°–39°C). Symptoms precede the skin lesions, which are called syphilids.

These vary greatly and no one case is exactly like another. The skin lesions vary in appearance and character, imitating many diseases, although they are never vesicular or bullous in acquired syphilis. (In early congenital syphilis, they may be.)

Lesions first to appear are macular, or maculo-papular, and are the commonest seen.

Early Lesions

Macular
Maculo-papular
Papular

Late Lesions

Annular
Pustular
Psoriasiform

} All uncommon

Macular lesions (Fig. 30)

vary in size from 0·5 to 2 cm, and are round. Their colour is rose or red and may only be seen in natural light and at a distance. Sites commonly affected are the shoulders, chest, back and arms. They may persist for only a few days, or change into papular lesions.

Papular lesions

develop from macules, or appear spontaneously as the first sign of the secondary stage. Similar in shape and size to macular lesions, the colour is coppery. Sites commonly affected are trunk, arms and legs, palms (Plate 17), soles and face. On the hair-line of the forehead and temples, papules may congregate like a garland, which has been called caustically the *corona veneris*. They may be scaly and, above all, are definitely indurated when palpated.

Moist papular lesions also occur, better known as *condylomata lata*. They are shiny, fleshy, flat, firm or soft, poorly or well-defined lesions. The sites affected are the ano-genital, axillary and sub-mammary areas, between the fingers and toes, and the angles of the mouth.

Moist papular lesions on the mucous membranes are known as *mucous patches*. They are about half an inch across and are irregular in shape; in the pharynx they are long, narrow and eroded and called snail track ulcers. The surface of the lesions is covered with a greyish film, which can be scraped off, leaving a pink area which does not bleed. These moist papular lesions are prolific with spirochaetes.

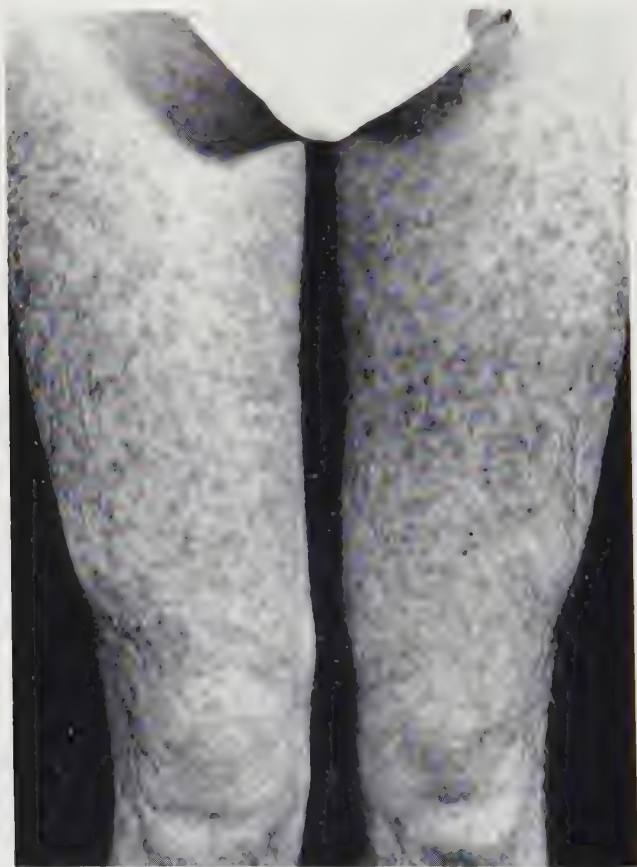


FIG. 30. Secondary syphilis: macular lesions (Institute of Dermatology, University of London).

Other signs are

1. Alopecia, which is not common, but when it does occur is patchy, involving the scalp, beard and eyebrows.

2. Jaundice may occur in severe infections.
3. Secondary anaemia, and a leucocytosis of 10,000–20,000 per ml with 50–80 per cent lymphocytes.
4. Generalized lymphadenopathy.
5. Atrophy of the skin where lesions have healed may occur.

DIAGNOSIS

This is made by finding the *Treponema pallidum* from the skin or mucous membrane lesions, and a positive W.R. and P.P.R.

Macular syphilis must be differentiated from the *acute exanthemata* such as *measles*, *drug eruptions* and *pityriasis rosea*; papular syphilis from *psoriasis*, *seborrhoeic dermatitis*, *urticaria*, *lichen planus*, and *drug eruptions*; pustular syphilis from other pustular conditions, including *bromide* and *iodide eruptions*; mucous patches from *Vincent's angina*, *tonsillitis*, *aphthous ulcers*, and *lichen planus*.

TERTIARY STAGE

This occurs 2–10 years after infection. Patients can often give no history of a primary or secondary stage. No organisms are found in the lesions. Lesions are all gummatous. There are three types: (1) nodular, (2) squamous, (3) single gummata.

SIGNS

Nodular lesions vary in size from a pin-head to a pea. They are reddish and well defined. Distribution is assymmetrical. Squamous gummatous lesions may be large, spreading across the body. Their shape is variable, being polycyclic or arciform and well defined, with activity apparent on the edge.

Single lesions start as smooth painless swellings which break down to form ulcers, which look as if they had been produced by a metal punch. In time they may involve underlying tissues. These particularly attack the leg, scalp, face, and sterno-clavicular areas. They may also attack the mucous membranes of the mouth, throat, and nose, being diffuse or localized.

Other signs are

1. Bursitis of elbow joint, acromial process of the scapula, and extensor tendons of the fingers.
2. Cardiovascular, C.N.S., and visceral complications.

DIAGNOSIS

is made by the history, and serological tests. These tests are positive in 90 per cent of cases, but a negative test does not exclude syphilis. C.S.F. examination should be done in all cases in the tertiary stage.

Tertiary syphilis must be differentiated from *lupus vulgaris*, from *leprosy*, and *carcinomata*.

TREATMENT

Prophylactic

1. Education of the public, though not enough of this is done.
2. Condoms, and they may not afford complete protection.

Curative

All early cases are given 600,000 units of Penicillin intramuscularly daily for 10 days, and this also applies to all late cases, with the exception of neurosyphilis which should be given the same doses for three weeks.

Congenital Syphilis

As already stated, infection is contracted from the child's mother. Paternal transmission from the semen does not occur.

Prematurity and death are common at birth. The signs of congenital syphilis usually occur within a month of birth, but in rare cases, are delayed for 15–20 years.

New cases of congenital syphilis are rarely seen now because of routine ante-natal W.R. and P.P.R. tests in this country.

CLINICAL FEATURES

In the early stages, these are coryza, nasal discharge, snuffles, wasting and insomnia.

Lesions may be macular, papular or bullous. When bullae break, ulcers form. Any site may be affected, but the buttocks, the palms and soles are invariably attacked. The bullae contain spirochetes.

Linear fissures may occur at the angles of the mouth and healing of them in later life appears as rhagades. Peri-anal condylomata appear a little later; infants in this phase are extremely contagious.

Other signs are the saddle-nose, saucer-shaped facies, sometimes painful and not always symmetrical hydrarthrosis (Clutton's joints), Hutchinson's triad of interstitial keratitis, deafness and notched incisors of the second dentition, and other signs reflecting involvement of other organs.

TREATMENT

Curative

600,000 units of penicillin for 10 days, and if there is evidence of neurosyphilis, a 3-week course is necessary.

Tuberculosis

Tuberculosis, once the scourge of humanity and the tragic source of pathos in novel and opera, has declined sensationally, at any rate in the Western Hemisphere. The improvement has been partly due to the introduction of effective chemotherapy.

Tuberculosis of the skin is now a rare disease. It exists in two principal forms.

I. Localized

- | | |
|----------------------------|--------|
| (a) Lupus vulgaris | |
| (b) Chancre | |
| (c) Tuberculosis verrucosa | } Rare |
| (d) Tuberculous gumma | |

II. Haematogenous forms, known as tuberculids

- | | |
|------------------------|--------|
| (a) Papulo-necrotic | |
| (b) Erythema induratum | |
| (c) Lichenoid | } Rare |
| (d) Acne agminata | |

Lupus Vulgaris

In this type, the tubercle bacillus is directly inoculated into the skin, or reaches it by spreading from underlying infected glands or joints, or via the lymphatics from an upper respiratory infection. It is commoner in women than men.

PATHOLOGY

The tubercles typical of tuberculosis with epithelioid and Langhans cells, lymphocytes, plasma cells and caseation can be seen in the dermis. In the healing stage, extensive fibrosis is apparent.

CLINICAL FEATURES

The onset is insidious. First lesion is a deep nodule, slowly forming a plaque as it coalesces with neighbouring lesions. The lesion may ultimately ulcerate, or atrophy, or hypertrophy. The shape of the lesion is always irregular, and the surface may be covered with an adherent crust. The colour is yellowish red, but when a watch-glass is pressed on it (this examination is called diascopy) the lesion becomes white, and the nodules appear as brown apple-jelly-like spots in the centre. The sites commonly attacked are the nose, cheeks and ears. In pre-chemotherapeutic days, the nose was destroyed. Large lesions can also be found on the trunk, hands and feet, in some cases. The buccal mucous membrane lesions appear as grey granulomatous patches; the tongue when attacked has large painful fissures.

Complications

Carcinoma *in situ* occurs in 1 or 2 per cent of cases.

DIAGNOSIS

This is made by the indolence of the lesion, the 'apple-jelly nodules', the tuberculin patch test and biopsy. The disease must be distinguished from *syphilis*, in which lesions are progressive, from *lupus erythematosus* whose lesions are covered by tiny horny plugged scales, *psoriasis* which shows heaped-up scales and involvement of the extensors, and *sarcoidosis* in which lesions evolve far more rapidly and are multiple.

TREATMENT

Isoniazid (Iso-nicotinic acid hydrazide) is the drug of choice. 300–400 mg daily for 6 months are given, and then for 3 months after cessation of all activity. Toxic effects are rare and disappear on reducing the dose, or stopping the drug.

Isoniazide is usually combined with Streptomycin or Para-amino-salicylic acid. Toxic erythemas and tinnitus are common side-effects.

Papulo-Necrotic Tuberculid

Tuberculid means a tuberculous condition in which the presence of tubercle bacilli cannot be demonstrated, although the pathology of the lesion resembles tuberculosis. This type of tuberculid, as the words imply, means a condition presenting a papule with (central) necrosis.

They are found in young people and appear in crops as small round discrete lesions on the exterior surfaces of the arms and legs. On healing, scars will be found.

TREATMENT

The same as for lupus vulgaris.

Erythema Induratum

Bazin's Disease

This condition is characterized by symmetrical red indurated nodules on the backs of the legs, and occasionally elsewhere. It predominates in females, between the ages of 10 and 20.

PATHOLOGY

This shows a non-specific or tuberculoid infiltrate in the lower part of the dermis. Proliferative changes occur, and caseation which accounts for the clinical breakdown of the lesions.

CLINICAL FEATURES

The lesions are notably painless.

The nodules break down, ulcerate and leave scars. The edges of the ulcers are steep or undermined. The lesions may persist for months.

DIAGNOSIS

The indolence, symmetry and painlessness of the lesions distinguish this disease from *erythema nodosum* which is a painful condition, without ulceration, *nodular syphilid* which will reveal other signs of the disease, and *chilblains* which are tender, and rarely ulcerate.

Treat as for lupus vulgaris.

Tuberculosis Verrucosa

This is a warty condition resulting from accidental inoculation whilst handling infected materials from autopsies, or meat. It is also known as a prosector's wart.

Anthrax

This rare condition is also known as a malignant pustule, but is only occasionally fatal. It is an acute infection caused by the anthrax bacillus, and characterized by a gangrenous carbuncular lesion, accompanied by constitutional symptoms.

PREDISPOSING CAUSES

As anthrax is a disease of animals, cattle-men, woolsorters, tanners and butchers carry the occupational risk of contracting it.

The disease may also be contracted through the hair of shaving brushes.

PATHOLOGY

The epidermis is destroyed and replaced by an ulcer on which bacilli can be found in large numbers.

CLINICAL FEATURES

Incubation period is 12–72 hours. Headache and high fever are followed, if untreated, by prostration and delirium. A small red papule, about the size of a flea-bite, becomes progressively vesicular, pustular and bullous, and then breaks down.

DIAGNOSIS

is made by isolating *B. anthracis*, and thus distinguishing the condition from a *carbuncle*, or a *chancre*.

TREATMENT

Penicillin or tetracyclines are effective orally.

PROGNOSIS

This is good when an early diagnosis has been made, otherwise poor.

CHAPTER 10

Fungal Diseases

Introduction

Fungi are non-photosynthetic plants, and do not contain chlorophyll. They depend on the tissue in which they exist for growth. Many hundreds of fungi abound, yet only a few are capable of producing disease. Those capable of doing so are divided into the superficial and deep species, depending on whether the resulting condition is associated or not with a systemic disease. In this chapter, reference will only be made to the superficial type of fungal infection.

The number of cases of fungal diseases cannot be categorically stated as it varies from one part of the world to another, but in the British Isles it usually accounts for 5 per cent of all cases of skin disease.

Superficial fungi live *on* the skin, and nearly entirely on dead horny tissue, living structures usually being avoided. They digest keratin, the structural basis of the horny layer of the skin, the nails and hair, resulting in disintegration of these structures.

The fungi consist of two parts: (1) the mycelium or vegetative part, which consists of filaments or hyphae and acquires food; (2) the spore, or reproductive part, which is a mass of protoplasm surmounted by its wall.

There are three families, or genera, of superficial fungi: *Trichophyton*, *Microsporum*, and *Epidermophyton*. The diseases they cause are not very infectious nor very contagious, and contraction of the disease depends to a great extent on individual susceptibility and predisposing factors such as hyperidrosis.

Some fungi also cause disease which produces symptoms of cutaneous and/or systemic involvement. Examples of such conditions are actinomycosis, blastomycosis, and histoplasmosis; descriptions of such disorders will be found in larger textbooks.

Tinea

The most important superficial fungi are the ringworm fungi which attack the skin, nails and hair (Fig. 31).

The *diagnosis* of tinea is made by microscopical examination of scrapings from the lesions, which have been placed in 10 per cent potassium hydroxide.

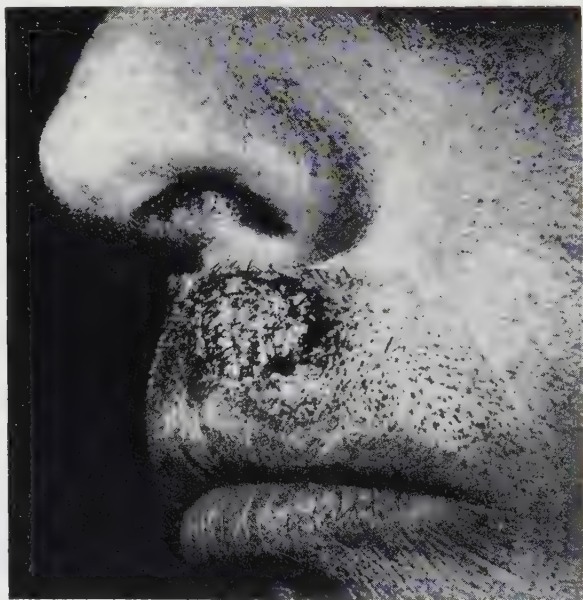


FIG. 31. Tinea barbae. Involvement of the moustache area with *T. discoides*, which responded well to griseofulvin.

Culture of the scales should also be done, as microscopy may sometimes fail to reveal the fungus. The species of fungus may be identified by culture. In ringworm of the scalp in children, hairs may fluoresce under Wood's light, depending on the species present. Wood's light is a mercury vapour lamp, containing cobalt and nickel oxide in a glass filter. Normal hair and skin fluoresce slightly under this light with a blue-white colour, but hairs infected with some types of ringworm fluoresce with a remarkable greenish colour. It may be used for the diagnosis and control of ringworm of the scalp.

TREATMENT

The treatment of ringworm was revolutionized in 1959 by the introduction of griseofulvin. It is an antibiotic, and is obtained by the fermentation of several strains of penicillium.

It is effective against all known species of *Trichophyton*, *Microsporum*, and *Epidermophyton*.

It is *not* effective against *Candida albicans*, or any fungi causing systemic conditions.

Toxic reactions are rare; those reported include nausea, vomiting, headaches and urticaria. For contra-indications to griseofulvin therapy see page 34.

Resistance to the drug is also very rare.

Griseofulvin is taken by mouth. The average daily dose is 1.0 g daily, and the dose may all be taken at the same time. Recent preparations of griseofulvin tablets, each containing 125 mg in fine particles given in doses of 500 to 1,000 mg daily, are equally effective. Griseofulvin has superseded all other forms of treatment. When, however, only one or two lesions exist, a fungicidal ointment may be used. The exception to this rule is in cases of scalp infections when griseofulvin must always be used.

Tinea pedis

Athlete's foot, ringworm of the feet

This condition is found mostly in young and middle-aged men, more commonly in summer than winter, and rarely in children. There are two types, namely an acute inflammatory, and a chronic dry. The organism usually found in the acute type is *Trichophyton mentagrophytes*, and in the chronic type *Trichophyton rubrum*.

CLINICAL FEATURES

1. *Acute Inflammatory*

This type arises from what has been for months or years a minimal infection, characterized by symptomless scaling in the toe-clefts, usually between the 4th and 5th toes. Suddenly an acute vesicular or vesiculo-pustular eruption begins which may spread over the feet and legs. Itching is common.

2. *Chronic dry*

This type is clinically mild, yet usually very difficult to cure. It is characterized by slight redness and scaling (Fig. 32), involving the soles, heels and sides of the feet (moccasin appearance). Acute exacerbations are uncommon. The nails invariably become involved, the changes being thickening under the nail, and friability of the nail.



FIG. 32. Tinea pedis showing well-marked scaly edge. Causative fungus, *T. mentagrophytes*.

DIAGNOSIS

Clinical signs of ringworm of the feet are not reliable and must be substantiated by microscopical examination of scrapings.

The condition must be differentiated from *contact dermatitis* caused by shoes, when the areas of contact with the shoes should be evident, *hyperidrosis*, which shows simple maceration between the toes, and in the chronic type of ringworm, from *psoriasis*, whose lesions show well-defined plaques.

TREATMENT

In the *acute* phase of the *acute type*, treat only with bland applications.

Complete or partial rest is very rewarding. Blisters should be opened, but not removed.

Potassium permanganate warm foot baths, 1 in 8,000 solution, should be given for 5 minutes two or three times a day.

Griseofulvin is not very helpful in this phase.

As the condition becomes subacute, or chronic, apply half-strength Whitfield's ointment twice daily, or freshly-prepared Castellani's paint. Griseofulvin may now be given.

For the *chronic dry type*, apply Whitfield's ointment or Castellani's paint, and give griseofulvin 1.0 g daily, although, as already stated, results are often disappointing.

Tinea Manuum

Ringworm of the hands

As in ringworm of the feet, there are two clinical varieties, an acute and chronic, the acute being very uncommon in temperate climates. The fungi responsible for both varieties are invariably *Trichophyton rubrum*, or *T. mentagrophytes*.

CLINICAL FEATURES

These are similar to foot infection.

This condition hardly ever exists without coincidental involvement of the feet, and they should therefore always be examined, although the patient usually denies that there is anything wrong with them. Examination of the feet, in fact, should never be omitted when a rash exists on the hands.

DIAGNOSIS

This must be made by mycological investigation and from clinical signs regarded as corroborative rather than diagnostic evidence. Ringworm of the hands is not very common.

TREATMENT

This consists of giving 1 g of griseofulvin daily, yet even after apparent cure has taken place, relapses occur in a notable percentage of cases. Fungicidal ointments may have to be used at the same time.

Tinea Unguium*Ringworm of the nails*

The fungus commonly responsible for this condition is *Trichophyton rubrum*. *T. mentagrophytes*, *T. interdigitale*, and *E. floccosum* are much less common. Invariably ringworm of the feet is present.

CLINICAL FEATURES

Every degree of nail deformity may be seen, from one scarcely detectable to virtually complete disintegration of the nail plate (Fig. 33).



FIG. 33. Tinea of nail, *T. rubrum*.

The infection begins at the distal end of the nail plate and spreads proximally. The undersurface of the nail may be seen to be hyperkeratotic.

The colour of the nail does not alter unless bacterial invasion takes place, when it becomes greenish. Nevertheless, there is ordinarily a loss of the normal lustre of the nail.

DIAGNOSIS

This is made by microscopical and cultural examination of the nail clippings. The condition must be distinguished from *psoriasis*, which nearly always shows typical psoriatic lesions on the characteristic sites. *Dermatitis* of the fingers from any cause is liable to produce nail changes similar to fungus infection.

TREATMENT

Griseofulvin 1 g daily must be taken for many months before a cure is obtained; on an average 6 months for fingernails and 18 months for toenails. In some cases avulsion has also to be considered.

Tinea corporis; Tinea Circinata

Ringworm of the body

Practically all species of trichophyton and microsporum are capable of involving any area of smooth skin.

CAUSE

Microsporum canis, *T. mentagrophytes*, *T. rubrum*, and occasionally *T. tonsurans* and *T. verrucosum* are the most important of the others.

Children are more susceptible than adults.

CLINICAL FEATURES

Four common varieties of lesions are seen, each usually being associated with a particular type of fungus.

The varieties are (1) annular, (2) plaque, (3) follicular, and (4) granulomatous.

1. The *annular type* produces red-ringed lesions, with tiny peripheral vesicles, and a clear, slightly scaly centre. The scalp may also be involved.

2. The *plaque type* is less inflammatory than the annular type, and may also involve the scalp. *T. tonsurans* is the normal organism found. Occasionally *T. rubrum* produces large plaques.

3. The *follicular type* shows a pustular folliculitis, usually of the neck, shoulders and arms, which may be quite painful. Granulomatous lesions sometimes occur. The source may be traced to pet white mice which may have been permitted to run up and down the arms of children. The organism is invariably *T. mentagrophytes*.

4. The *granulomatous type* is characterized by lesions looking like carbuncles. Suppuration is present and is due to the fungus, usually *T. verrucosum*, which also causes cattle ringworm, and can be transmitted from animal to patient. In this case, too, the scalp may be involved.

Other varieties exist, but they are all rare.

DIAGNOSIS

This is made by microscopy and culture when necessary. Wood's light is positive in some cases when the scalp is involved.

The disease must be distinguished from *pityriasis rosea*, in which no fungi are found, and evolution is much more rapid. *Psoriasis* has heaped-up scales, nearly always on extensor surfaces, and vesicles are absent. *Eczema* shows no central clearing. *Seborrhoeic dermatitis* has greasy scales involving seborrhoeic areas, and the lesion is not clear in the centre.

TREATMENT

Topical ointments are usually all that is needed, Whitfield's half strength to start with usually being effective. If this fails, griseofulvin must be given.

Tinea Cruris, Dhobie Itch

Ringworm of the Groin

This condition occurs predominantly in males. The organisms responsible are the same as those producing ringworm of the body. The condition is bilateral, but asymmetrical, and annular type lesions are produced (Plate 18). Itching may be quite marked.

TREATMENT

Griseofulvin is best. Topical therapy is messy.

Tinea Capitis*Ringworm of the scalp*

This condition is characterized by scaly patches of different sizes in which broken hairs may be seen. It occurs nearly exclusively in children (Fig. 34, Plate 19).

CAUSE

Little of the anthropophilic variety is now seen in the British Isles. Animal types may be seen in country areas. *T. verrucosum* is still quite common. *M. canis* is rare.

CLINICAL FEATURES

The onset varies according to the fungus involved. Generally, however, it is gradual. The inflammatory reaction in the patch also depends on the fungus present. If the condition is allowed to persist untreated, a folliculitis, or a carbuncular mass called a kerion, develops. As a result of modern methods of treatment, this is now rare.

The disease may spread to the eyelids, the neck and trunk, and these areas must be examined.

A special aid to diagnosis is the use of Wood's light (see p. 121). In most cases of ringworm of the scalp, fluorescence occurs, but some fungi do not cause it.

DIAGNOSIS

This is made by the clinical appearance of scaling and broken hairs, and microscopical examination of hairs in potassium hydroxide.

The condition must be differentiated from *seborrhoeic dermatitis* in which there are no broken hairs and greasy scales are present. *Alopecia areata* shows no scales, and the scalp is white, shiny and smooth.

TREATMENT

Griseofulvin is the drug of choice. 1·0 g daily for a month nearly always cures; 3–4 g can be given as a single dose.

For children, one single large dose of 3.0 g is usually effective and can be repeated at the end of a month if no improvement has been noted.

Whitfield's ointment should be applied daily to affected areas, if only to prevent debris falling. Starch poultices may also be required.



FIG. 34 Tinea capitis: causative fungus, *T. mentagrophytes* (Institute of Dermatology, University of London).



PLATE 20. Moniliasis.
Polycystic borders
fringed by white
epidermis
(Dr Martin Beare).

PLATE 21. Molluscum
contagiosum.
Pearly lesions on the
back, showing the
central depression from
which a caseous
plug may be squeezed.

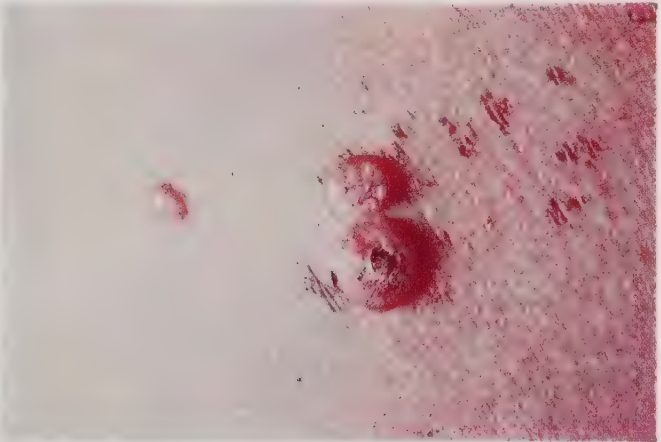




PLATE 22. Xanthomatous nodules on a boy with a high blood cholesterol and a family history of the disease.



PLATE 23. Lupus erythematosus: chronic variety (Institute of Dermatology, University of London).

Tinea Versicolor*Pityriasis Versicolor*

This is a chronic, symptomless fungus infection due to *Malassezia furfur*, which appears in scales as hyphal elements and spores.

CLINICAL FEATURES

The onset is gradual. The lesions are macular and are fawn or café-au-lait. They are well defined, covered with fine branny scales, and they commonly affect the chest, back and axillae. Those affected usually wear woollens, often sleep in them, and bath seldom.

DIAGNOSIS

This is made by finding the fungus in scales. The infection must be distinguished from vitiligo, chloasma, and other pigmentary disorders.

TREATMENT

Frequent washing and change of underclothes which should be cotton. Apply Whitfield's ointment daily, and for 2 weeks after signs of infection have disappeared.

Moniliasis*Candidiasis, thrush, oidiomycosis*

Moniliasis produces a variety of lesions of the skin and mucous membranes, caused by a yeast-like fungus, *Candida albicans*.

PREDISPOSING FACTORS

Occupation: housewives, bar-tenders and bakers are prone to develop paronychia. Excessive immersion in water allows the organism to enter below the softened nail-fold.

Other illnesses: Debilitating diseases, alcoholism, diabetes, hyperidrosis and obesity may predispose to monilial infection.

CLINICAL FEATURES

Localized types

(1) Onychia and paronychia. These conditions are characterized by bolstering of the affected nail-fold from which, perhaps, a bead of pus may be expressed with gentle pressure. The nail is usually ridged as a result of the disease, and a dirty greenish-brown discoloration of the lateral aspects of the nail takes place.

(2) Intertrigo. This is characterized by well-defined red, moist patches with a scalloped edge, commonly in the submammary folds, groins, umbilicus, axillae, or intergluteal folds (Plate 20).

(3) Perlèche. See chapter 18.

(4) Intra-oral thrush. This is seen as a whitish loose membrane on the inner surface of the cheeks, or on the palate of babies, children or young adults.

(5) Superficial glossitis. This may appear in adults as a beefy-red, smooth tongue.

(6) Vaginitis, vulval ulcers, and balanoposthitis.

Generalized types

These may be cutaneous or systemic, but are uncommon.

DIAGNOSIS

This can be made by finding the organism in scrapings from lesions.

TREATMENT

For paronychia, avoid soap and detergents completely, using finger-stalls or rubber gloves. Apply nystatin ointment 2 or 3 times a day, or eusol lotion, by inserting the application under nailfold with a flat orange stick.

For intertrigo, use nystatin ointment, or ichhammol powder. Amphotericin B, 2 or 3 per cent in ointment form, may also prove useful.

For thrush, use nystatin 100,000 units per c.c. of water, and compound thymol glycerin B.P.C. mouth washes, t.d.s.

CHAPTER 11

Diseases due to Viruses

Introduction

Viruses are very minute organisms, smaller than bacteria and not visible by the usual microscopic examination. They cannot carry on a free life of their own like bacteria, and live intracellularly, breeding and multiplying in living cells.

They tend to have certain fixed habits, which are useful for identification purposes. They usually focus their attention on one particular kind of host and one particular kind of tissue and produce in that tissue particular types of intra-cellular inclusion bodies.

The diseases which are described below are considered to be viral in origin, and this is substantiated by the distinctive inclusion bodies which can be found when the tissue has been appropriately stained.

In herpes zoster and simplex they are called Lipschütz bodies, in molluscum contagiosum, molluscum bodies, in varicella and variola, Guarnieri bodies. All diseases due to viruses are infectious or contagious, though some more mildly so than others.

Warts

Nearly all warts, or verrucae, are of the common type and are viral in origin. Other less common varieties are (1) syphilitic, (2) malignant and (3) tuberculous which are dealt with elsewhere.

Common warts are the bane of the dermatologist's practice. They may form a third of hospital cases, they are by no means straightforward to treat successfully when large numbers are present, and are liable to recur. They also appear on new areas following eradication of existing lesions. Some individuals are more susceptible than others, including the author, and it is noticeable that in a household or in a



FIG. 35. Penile warts (Institute of Dermatology, University of London).

school, where contacts are closer than normal, many remain immune to this infection—a characteristic of virus diseases.

Warts are well-defined tumours of varying size, having a rounded or pointed top and a sessile or pedunculated base. There are several

clinical forms of the common wart: (1) the common, (2) the plantar, (3) the plane or juvenile, (4) the filiform, (5) the acuminate.

PATHOLOGY

Hyperkeratosis, acanthosis, and papillomatosis are the principal changes seen in warts, and these hypertrophic reactions account for the density of warts. Inclusion bodies are present in some, but not all warts.



FIG. 36. Common warts.

CLINICAL FEATURES

(1) *The common wart (Verruca vulgaris)*

Lesions are pin-head to pea-size. Sites: hands and fingers chiefly (Fig. 36).

(2) *The plantar wart (Verruca plantaris)*

is pea-sized or larger and may resemble a corn (Figs. 37, 38). Pressure points on the plantar surface of the foot are chiefly affected, but no part is exempt.



FIG. 37. Plantar wart of ten years' duration, having weathered many different local applications, and some operative procedures.

(3) *The plane wart (Verruca plana)*

so-called because they are flat-topped. They are very discrete and flesh-coloured and, if single, may be difficult to observe. Sites: the face, forehead, the backs of the hands and the front of the knees are most commonly attacked. Young people are predominantly affected.



FIG. 38. Plantar wart, mosaic variety.

(4) *The filiform wart (Verruca filiformis)*

is pedunculated and may be as thick as a match and up to a quarter of an inch long. It is commonly seen on the necks of middle-aged women and the bearded area of men.

(5) *The acuminate wart (Verruca acuminata)*

is soft and fleshy and resembles a miniature cauliflower; with time they vegetate and stink. They develop on mucocutaneous areas, especially the ano-genital.

TREATMENT

There is no specific treatment for warts. Any form of conservative or radical treatment may succeed or fail. One physician will succeed with an identical treatment with which a colleague has failed. Such is the fickle nature of warts. A large number of warts, probably 5 per cent, disappear spontaneously, and many can be tempted to leave their host by remedies as varied as the application of early morning spittle, the eye of a potato or sticking-plaster, providing the practitioner can convince the patient of their value.

Common warts

40 per cent salicylic acid in collodion, or 5 per cent picric acid may be applied daily. Cauterization under local anaesthetic or curettage is the most effective treatment and most useful when few warts are present. The application of liquid nitrogen or oxygen is an impressive form of therapy, but painful and not always effective.

Plane warts

Invariably respond to local applications.

Plantar warts

Are best treated by cautery or curettage. Otherwise formaldehyde 1 per cent in water applied daily for 5 minutes on cotton wool may be curative after 4–8 weeks. The mosaic variety may be treated by weekly applications of 40 per cent salicylic acid plasters, kept in position by sticking plaster.

Filiform warts

Are easily cauterized.

Acuminate warts

usually respond to 25 per cent podophyllin in alcohol or tinct. benz. co. applied carefully every 2 or 3 days for 3 or 4 applications. If the application is placed on normal skin, it causes a painful burn. If it fails, cautery is the treatment of choice.

PROGNOSIS

Recurrences are by no means uncommon.

Herpes Zoster*Shingles*

This is an acute infection of nerve structures producing groups of vesicles distributed along one or more peripheral nerves on one side of the body. The inflammatory changes are usually confined to one posterior root ganglion.

CAUSE

The virus is either identical with or closely related to the virus of chicken-pox, since it is not uncommon for healthy contacts with either disease to contract the other.

Age

Any age may be affected, but it occurs chiefly in adults.

Season

As in so many viral diseases, infection is more likely during the spring and autumn.

Secondary to other conditions

Less commonly the eruption appears following trauma, pressure from vertebral tumours, or the enlarged glands of Hodgkin's disease or leukaemia, or infections such as meningitis.

PATHOLOGY

The vesicle is intra-epidermal and is produced by degenerating epidermal and blood cells and fibrin, which it also contains.

The epidermal cells are apparently inflated, appearing as balloons. They stain poorly, and may be un-nucleated, or contain many nuclei. In the basophilic nuclei of the balloon cells are found the eosinophilic Lipschütz inclusion bodies, and the epithelial cells of the hair follicles and sebaceous glands may be similarly affected.

Another notable change is reticular degeneration, which means cavity formation in the epidermis, resulting in multilocular vesicles. Reticular degeneration, it must be remembered, occurs sometimes in dermatitis. The dermis shows generalized vascular dilatation, a polymorphic and lymphocytic infiltrate, and oedema in the upper third of the dermis.

The posterior nerve root ganglia show interstitial inflammation, and degenerative changes.

CLINICAL FEATURES

Pain of varying degrees of severity in the distribution of the root or roots affected precedes the vesicular eruption by a day or two. In the appendix area, for example, the pain may cause considerable difficulty in diagnosis before the rash appears. Mild pyrexia and fairly severe irritation may also occur.

The lesions are vesicular containing clear fluid and develop from papules. After a few days, the vesicles rupture and crusted lesions form. Occasionally the lesions are pustular or haemorrhagic. Their size is tiny or pea-sized. The site may be anywhere on the body and the distribution is nearly always unilateral (Fig. 39).

Other varieties

(1) Ophthalmic or Gasserian herpes involving the 1st division of the Vth cranial nerve is always serious, leading sometimes to keratitis or blindness.

(2) Geniculate herpes (the Ramsay-Hunt syndrome) commences with tonsillar or ear pain, and is followed by a vesicular eruption on the pinna, external ear, or fauces, loss of taste of the anterior two-thirds of the tongue, and a facial palsy of lower motor neurone type.

Complications

Post-herpetic neuralgia occurs especially in old people, and may be so distressing and chronic as to induce suicidal depression. It may, however, clear spontaneously in a few months.

DIAGNOSIS

This is made by the history, the grouping of painful vesicles, and the unilateral distribution. *Herpes simplex* is sometimes bilateral and commonly recurrent, but the first attack may be difficult to distinguish from zoster.

TREATMENT

There is nothing specific. Rest is paramount for the elderly.

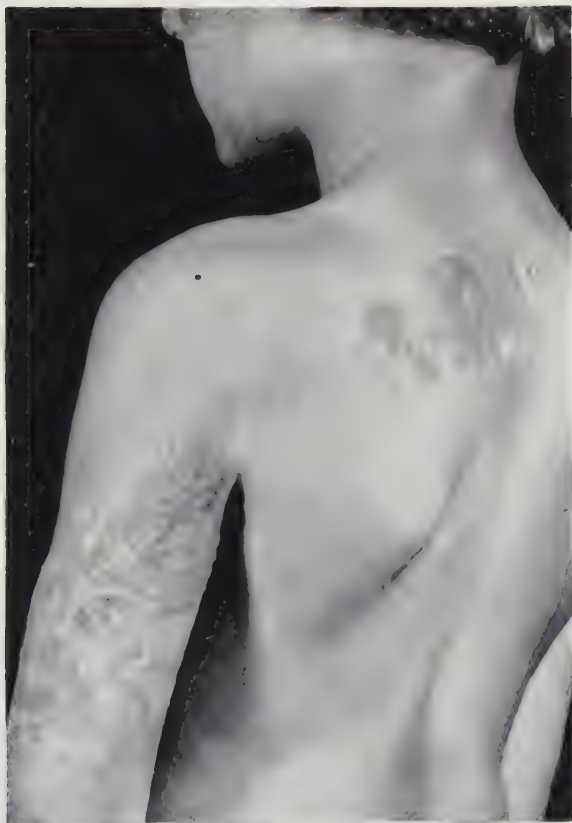


FIG. 39. Herpes zoster showing vesicular and unilateral eruption (Institute of Dermatology, University of London).

External

Calamine lotion with 2 per cent phenol, or a steroid lotion with an antibiotic should be dabbed on 3 or 4 times daily. The lesions should be protected from trauma with gauze.

Internal

Aspirin may be given to relieve pain.

Post-herpetic neuralgia usually offers a great challenge to ingenuity. Radiant heat to the healed area, X-ray to the affected ganglia, morphia, and steroids by mouth are some of the therapies which may be tried.

PROGNOSIS

This is better for young people than for old. Second attacks are uncommon.

Herpes Simplex

This is an acute disorder characterized by grouped vesicles on a red base, usually affecting the lips or face. The condition may be recurrent or non-recurrent, the former type being the rule. This disease presents a different picture from herpes zoster.

The word simplex in the title is extremely misleading, as the secondary type is notably recurrent, despite treatment.

CAUSE

A filtrable virus.

Predisposing factors may be strong sunlight, fevers, gastro-intestinal upsets, or emotional stress.

PATHOLOGY

This is similar to zoster.

CLINICAL FEATURES

Recurrent type. Unlike zoster, there is only slight discomfort when the lesions appear, the onset being sudden.

Lesions are vesicular at first, later rupturing and forming crusted lesions. Sites involved are chiefly the lips and face and also occasionally the genitalia and trunk. Lesions are grouped, as all herpetic lesions are, but differ from zoster in that they are sometimes bilateral.

COURSE

The lesions clear up in one or two weeks but are liable to recur, and when they do so, always attack the same area as before.

Other varieties

The *non-recurrent type* is seen in children between 6 months and 7 years of age. Pyrexia may reach 104°F (40°C). A large vesicle appears in the mouth, breaks down into an ulcer, and heals within a fortnight.

DIAGNOSIS

Herpes simplex must be distinguished from herpes zoster.

TREATMENT

There is nothing specific. Burow's lotion dabbed on 2 or 3 times a day, or eusol, keep the lesions clean. Tetracycline ointments are usually prescribed if the lesion becomes infected. A small dose of X-rays may shorten an attack, and lengthen the interval before the next one.

Kaposi's Varicelliform Eruption*Eczema herpeticum*

This is an acute, contagious, vesicular, febrile eruption, caused by the herpes simplex or vaccinia virus, superimposed on eczematous skin. It occurs predominantly in male infants and children.

CLINICAL FEATURES

The onset is sudden. Lesions are vesicular, occurring in crops on eczematous skin, which becomes oedematous. The attacks last 10-14 days.

DIAGNOSIS

is made by history of exposure to herpes simplex and the identification of Lipschütz inclusion bodies in early vesicles.

TREATMENT

Isolation, and antibiotics to counteract secondary infection.

PROGNOSIS

In severe cases, death may occur.

Molluscum Contagiosum

This disorder is characterized by rounded molluscous or shell-like lesions, showing on close examination an apical depression.

CAUSE

A virus, both auto-inoculable and contagious.

Predisposing factors

Age. Infants and children are most commonly affected.

Occupation. This disorder is not uncommon in beauty specialist and masseurs who contract it from their clients, and also transmit it to them.

PATHOLOGY

The epidermis develops into large pear-shaped lobules, extending itself into the dermis. Many epidermal cells degenerate and desquamate, and because of this, a cavity forms on the surface producing what is seen clinically as a central depression.

CLINICAL FEATURES

There are no symptoms. The onset is gradual. The lesions are papular at first, later becoming nodular (Fig. 40). Their size varies from pin-point to pin-head, and they are slow to develop. Their shape is slightly pointed or globular at first, later becoming flattened on the top, the surface being umbilicated. Their colour is pink or pearly (Plate 21) and they are shiny. Sites commonly involved are the face, eyelids, and genitalia, but no area is exempt. The number of lesions may vary from one or two to thirty or more. The contents of these lesions consists of a thick yellowish curdy substance.

COURSE

Untreated, the lesions persist for many months; sometimes some of them dry up spontaneously.

DIAGNOSIS

Once seen, the discrete, pearly, shiny, umbilicated lesions are unmistakable.

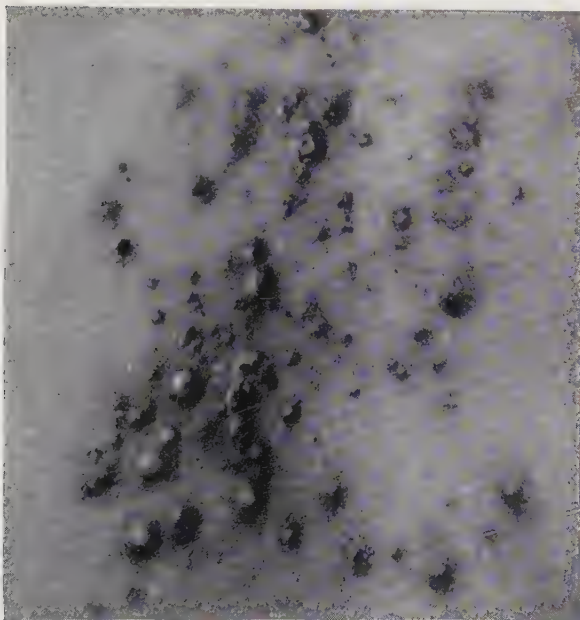


FIG. 40. Molluscum contagiosum. Pearly papules and nodules with some typically umbilicated.

TREATMENT

Lift off the crusted top, or incise the top of the lesion and express or curette the curdy contents. Then insert an orange-stick impregnated with pure phenol or iodine for a few seconds.

Chicken-Pox

Varicella

This acute infectious disease is characterized by crops of bright red vesicles, caused by a virus identical, or closely allied to, the virus of herpes zoster. It is most infectious in the early stages, but also infectious, though less so, while crusts remain.

CLINICAL FEATURES

Prodromal malaise may or may not occur. Pyrexia is usual, varying between 99° – 101° F (37.2° – 38.3° C).

Lesions appear on the first or second day. First of all papular, they soon become vesicular, and later pustular. Scars often follow resolution. Sites commonly affected are the trunk and face. The rash, unlike smallpox, is most profuse on the trunk, and sparsest at the periphery of the limbs. The mouth and throat also become affected. Duration of the disease is 3–7 days.

TREATMENT

is symptomatic. Calamine lotion is helpful for itching.

Smallpox*Variola*

This is an acute infectious and contagious fever, characterized by macules, papules, vesicles, pustules, crusts and scarring, with constitutional symptoms of varying severity.

Quite often, during epidemics, the dermatologist is asked to confirm a diagnosis of smallpox. Since vaccination is not compulsory the risk of epidemics is still considerable, and it is, therefore, as important as ever to be familiar with the different facets of the disease.

There are three varieties of the virus causing smallpox:

1. Asiatic smallpox (*variola major*), which has a high mortality rate.
2. *Variola minor*, which causes a far milder disease.
3. The third is used for vaccination, which cannot cause a serious disease.

No infant with eczema should be vaccinated against smallpox (see p. 42).

PATHOLOGY

Numerous Guarnieri bodies are found in the early stages. They are eosinophilic, round or oval, and are surrounded by a notably clear halo. Reticular degeneration is well marked, but unlike herpes zoster there are few balloon cells.

CLINICAL FEATURES

The incubation period of 12 days (in most patients) is followed suddenly by severe headache, pyrexia 103° – 104° F (39.4° – 40° C), pain in the back, rigors, and generalized aching of the limbs. A day or two after the appearance of the rash the temperature subsides, and the patient improves. This is a period when optimism is dangerous, for a few days later, as pustular lesions develop, the fever returns in full spate, and the patient becomes gravely ill again. If the patient recovers, the temperature slowly subsides again as the pustules dry up.

Prodromal purpuric lesions are seen in severe cases from the start. Lesions characteristic of smallpox appear on the third day as macules; within 24 hours they become papular, about three days later they become vesicular, and a few days afterwards, pustular. Three or four days later, crusts form, and in several weeks' time the crusts fall off. The sequence is therefore:

Macules—Papules—Vesicles—Pustules—Crusts.

All the lesions pass through this series of changes at the same time.

Sites first affected are the hands, wrists, forehead and mouth, mucosal lesions often being very severe. The rash spreads from the outer parts of the body towards the trunk, contrariwise to chicken-pox.

Complications

include broncho-pneumonia, conjunctivitis, otitis media, corneal ulceration with subsequent opacities, encephalitis and cardiac failure.

DIAGNOSIS

Sometimes difficult in mild cases to distinguish from chicken-pox. In chicken-pox there is far more involvement of the trunk, and the lesions appear in crops. Smallpox can be diagnosed with certainty by finding Guarnieri bodies in fluid from vesicles.

TREATMENT

There is no specific therapy. Skilled nursing is of the greatest importance. Aureomycin by mouth eliminates secondary infection.

Recently, trials with N-methylisatin P-thiosemicarbazone (Marboran) in the prophylaxis of smallpox have proved this drug to be invaluable in its prophylactic effect, regardless of the vaccination status of contacts.

Measles

This is an acute infectious and contagious disease, characterized by a macular rash, coryza, and upper respiratory catarrh.

Infection in the first 3 months of life is impossible, as a result of passive immunity conferred by the mother, but at any time afterwards the individual becomes highly susceptible. Transmission is by direct contact, from the nose, mouth, or respiratory tract. Infectivity lasts for 2 weeks after onset of the rash.

CLINICAL FEATURES

10–14 days after exposure, coryza, cough, and fever set in. The rash is always itchy.

Koplik's spots are diagnostic, and are visible on the first day. Koplik's spots are tiny white papules, like grains of salt, best seen on the mucous membrane of the cheeks opposite the molars.

On the 4th day, a pink macular rash which first appears behind the ears spreads over the face, trunk and limbs. The macules are small, being about 3–5 mm in diameter, but they coalesce into large irregular areas as the disease progresses, and finally fade as pale scaling lesions.

Complications

are principally due to secondary infection, resulting in bronchopneumonia, or otitis media.

DIAGNOSIS

Distinguish from German measles, which is much milder, and has no prodromal symptoms.

TREATMENT

Bed rest until the temperature has been normal for a week. Broad-spectrum antibiotics, such as tetracyclines, should be given to prevent bacterial complications.

German Measles*Rubella*

This is a mild infectious disease, characterized by a macular rash. When contracted by women in the first four months of a pregnancy, it often leads to developmental defects in the foetus. Transmission is by direct contact. Infectivity exists at all stages of the disease. Incubation period, 14–21 days.

CLINICAL FEATURES

Lesions are macular. Sites first affected—scalp and face, followed by a general spread, lasting 1–3 days. Occipital glands become enlarged and tender.

TREATMENT

is rarely required.

Vaccinia*Cowpox*

This is an eruptive disease occurring chiefly in the cow, but also produced in man by auto-inoculation or, very rarely, as a general infection.

One or more lesions occur on the body 7–14 days after vaccination. Ocular palsy and encephalitis are rare features of the disease.

Eczema vaccinatum describes vaccinia superimposed on eczema (see page 42).

Orf

This produces a contagious, pustular dermatitis in sheep or goats, and is contracted by those handling infected animals dead or alive. The incubation period of 3–7 days is followed by the appearance of a firm, painless, dark papule which enlarges to form a domed pustule which may contain a little serum or blood, but mainly granulation tissue. Lesions are usually single, and commonly found on the fingers and hands. The condition is self-limiting, clearing in 4–8 weeks. Mild local remedies are sometimes required.

CHAPTER 12

Bullous Diseases

Bullous disorders are not common and may be congenital or acquired.

The most important *congenital* form is epidermolysis bullosa, of which there are two varieties: a simple and a dystrophic type, the latter being characterized by lesions healing with severe scarring. A very rare lethal form also exists.

The most important *acquired* varieties of bullous eruptions are:

1. Dermatitis herpetiformis.
2. Pemphigus.
3. Pemphigoid.

Dermatitis Herpetiformis

This is a chronic condition characterized by vesicles, bullae, and papules, which, as herpetiform suggests, results in the lesions usually being grouped as they are in herpes zoster. Another factor of much more importance to the patient is the itching which always accompanies the condition and which may be most severe.

The cause is not known. Both sexes are affected at all ages, but chiefly adult males.

PATHOLOGY

The bullae and vesicles form below the epidermis, unlike pemphigus. An important feature whose absence or presence must be noted is that of acantholysis (see p. 10), which is absent in this disorder, and present in pemphigus. An eosinophilic infiltrate is another major sign, being obvious in the bullae and around blood vessels.

CLINICAL FEATURES

The onset is commonly gradual. The vesicular and bullous lesions are tense and clear at first, and later become cloudy. The groups of lesions have irregular patterns, and excoriations from scratching are notable, as the lesions resolve. The sites usually affected are the forearms and thighs, interscapular and lumbosacral areas, while the distribution of the rash is symmetrical. Mucous membranes are occasionally attacked. The general health is undisturbed, except when interfered with by insomnia through irritation.

Laboratory aids

The blood eosinophilia is high, being between 10 and 30 per cent. Patch tests to iodides are positive, and are carried out by applying a 20 per cent sodium or potassium iodide ointment to an affected area.

COURSE

It is typical of this disease to come and go, the intervals of relapse and remission varying between weeks and months, for up to 10 or 15 years, and very occasionally longer.

Other varieties

A juvenile type of dermatitis herpetiformis exists, which may start at infancy, and usually resolves at puberty.

TREATMENT

External

Calamine lotion, or a steroid lotion to alleviate symptoms slightly.

Internal

Diamino-diphenyl-sulphone (dapsone) is the drug of choice. 100 mg tablets twice a day for 2 weeks, followed by 100 mg daily or less, if less controls the disease, is given as long as is necessary. Iron should be given at the same time to offset any tendency to a normocytic anaemia, which the drug may provoke.

Sulphapyridine, 1-1½ g daily, has a similar effect, but may produce disturbing toxic effects. Steroids are of little help.

DIAGNOSIS

This is made by the itching, the grouping of the lesions, and their multiformity. For differential diagnosis, see end of this chapter.

Pemphigus

This is a chronic disorder characterized by recurrent bullae of the skin, and/or mucous membranes.

Before the introduction of steroid treatment it was fatal within a few months or years of its onset. The cause is unknown, and it is not infectious or contagious. Males are affected three times as often as females.

PATHOLOGY

The bullae form in the epidermis, in contrast to dermatitis herpetiformis, owing to degeneration and liquefaction of the epidermal cells. Acantholysis, in fact, is present. In scrapings from the floor of a fresh bulla, the degenerated cells can be easily found. They are round, and contain spherical nuclei. The cytoplasm of the cell is markedly clear, except for the edge of the cell, which appears as a definite dark blue ring.

CLINICAL FEATURES

The onset is insidious. The bullae are at first tense, round, and contain serum, which soon becomes purulent as the bullae become flaccid. The skin around the bullae is normal. The bullae soon rupture, and whether in the mouth or on the skin leave raw areas (Fig. 41), which are extremely tender, as crusts form. Their size varies, and they may be as big as a plum, whilst any site may be involved.

The patient's general condition deteriorates rapidly in accordance with the severity of the skin lesions.

Nikolsky's sign is invariably present. This means that when firm pressure with the finger is exerted on normal skin, the epidermis slides off owing to its generally poor attachment to the underlying dermis. This sign may be elicited in other severe bullous eruptions, such as those due to dermatitis herpetiformis, epidermolysis bullosa, or drugs.



FIG. 41. Pemphigus. 3 years' duration, with remissions (Skin Department, London Hospital).

Eosinophilia and leucocytosis sometimes occur. The lesions occur in waves, and sometimes quite long periods of remission occur.

Other clinical varieties

1. Vegetating pemphigus. A more serious and rare form.
2. Exfoliative pemphigus. Also rare.

3. Pemphigus erythematosus. Also uncommon, but usually benign, unless it slides into the true form of pemphigus. It suggests at the onset a combination of seborrhoeic dermatitis and lupus erythematosus.

DIAGNOSIS

This is made by the character of the bullae and acantholysis. At the onset it is difficult, without a period of observation, to differentiate it from other conditions it imitates.

For differentiation, see table at the end of this chapter.

TREATMENT

Steroid treatment can save the life of these patients, although death can occur in spite of this treatment. Huge doses may have to be given at first to improve the clinical condition, namely up to 200 mg of prednisolone daily until the dose can be gradually reduced. ACTH should also be considered.

The patient must be kept in bed for the preliminary study. Secondary infection must be avoided and antibiotics given when necessary.

Locally, the areas may be treated with potassium permanganate or eusol dressings, and in difficult and generalized cases, potassium permanganate baths are very useful.

PROGNOSIS

This depends greatly on the response to steroid treatment.

Pemphigoid

This fairly uncommon disorder is characterized by large bullae (Fig. 42), occurring in elderly people.

The cause is unknown, and it is relatively benign. The lesion is generally sub-epidermal, and therefore acantholysis is absent.

Steroids are invariably effective.



FIG. 42. Pemphigoid (Institute of Dermatology, University of London).

TABLE 2
Differential diagnosis of bullous eruptions

	<i>Dermatitis Herpetiformis</i>	<i>Pemphigus</i>	<i>Pemphigoid</i>
Age	Any	From 20 upwards	Over 60
Sex	Both	Both	Both
Eruption	Vesicles, and bullae	Bullae	Bullae
Site	Lumbo-sacral, buttocks, post. axillary folds common. Lesions are grouped	Face, neck, trunk, limbs. No grouping	Legs, arms, trunk
Mucous membranes	Rarely affected	Commonly affected	Not usually affected
Itching	Always present, and may be severe	Rare	Rare
Pathology	Subepidermal bullae No acantholysis	Intra-epidermal bullae Acantholysis present	Both types No acantholysis
Eosinophilia	10-40 per cent	Usual, but low	Unusual
Course	Chronic	Chronic	Chronic
Treatment	Dapsone	Steroids	Steroids
Prognosis	Disease lasts 5-20 years	Fairly good under steroid therapy	Quite good
*Iodide test	Positive	Negative	Negative

*The iodide test is carried out by applying a 20 per cent sodium or potassium iodide ointment for 24 hours to part of the eruption. If positive, the eruption is made worse.

CHAPTER 13

Diseases due to Parasites

Scabies

This is a contagious disease caused by infestation with *sarcoptes scabiei* (*Acarus scabiei*, Fig. 43), characterized by intra-epidermal burrows and follicular papules, and severe itching which is worse at night.

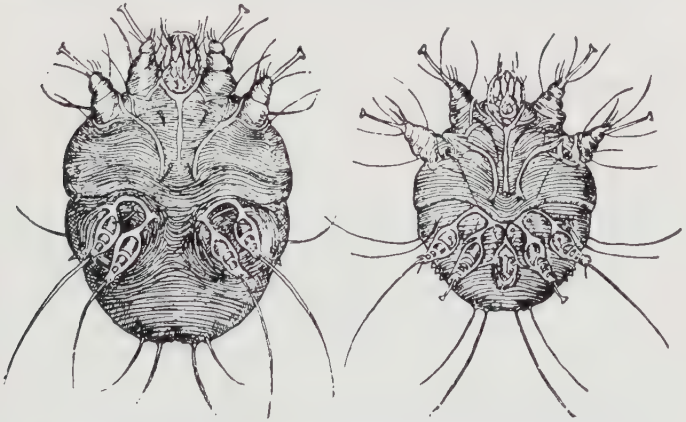


FIG. 43. *Acarus scabiei*, ventral surface. The female is the larger.

PARASITOLOGY

The female acarus is pearly grey, with 4 pairs of legs, is oval in shape and can be seen quite easily on the end of a pin, or on a glass slide. The male is seldom seen, as copulation proves to be fatal.

The pregnant acarus burrows into the horny layer of the skin into which she lays her eggs. The burrow is $\frac{1}{4}$ to $\frac{1}{2}$ an inch in length, with a roof that appears as a whitish zig-zag line, punctuated by one or more black specks which are caused by dirt or faeces in the burrow. The acarus may be found at the farthest end of the burrow by inserting a needle and gently breaking the roof at that point, followed by judicious probing which will cause the acarus to hang on the end of the needle.

The eggs hatch in 3–4 days, and six-legged larvae leave the burrow to find shelter in hair-follicles. They then slough their skin, twice, becoming eight-legged, and within two weeks adult females on impregnation initiate the cycle again.



50–100 eggs may be laid during the life of a female, during the course of 2 months.

Other varieties of scabies are horse, dog and cat scabies which may attack people in contact with them.

Transmission

of the disease is by close and prolonged contact. It is usually contracted in bed from an infested companion.

CLINICAL FEATURES

Remarkably severe itching occurs predominantly at night in bed, for that is the time when the person is warm and the mite prefers to promenade.

The onset is gradual. Lesions apart from burrows and papules also consist of pustules, excoriations, and crusted lesions. Sites of the burrows are the ulnar borders of the wrists, the palms, fingers, the anterior axillary folds, areolae of the nipples, lower abdomen, buttocks and penis (Fig. 44). Other lesions may occur anywhere. The skin above the neck-line is rarely, if ever, affected because the acarus avoids the cold as much as possible.



FIG. 44. Scabies: the intact part of the penile burrow can be seen below and to the right of the excoriated part. Burrows are more common on the hands, fingers, and ulnar border of the wrist (Dr G. A. Hodgson).

DIAGNOSIS

When lesions are minimal, scabies must often be consciously considered, and careful examination will reveal burrows. The social status of the person must never be taken into account for, like the flea, the acarus is not class-conscious. In well-developed cases, nocturnal itching and burrows are well established.

TREATMENT

Benzyl benzoate emulsion is applied following a hot bath, during which burrows are gently rubbed open with a soft brush; and a second application is given the following night without a bath. About two ounces of the emulsion are required for each application. Residual itching following this treatment should be dealt with by calamine lotion, and not more benzyl benzoate. All contacts should be similarly treated.

Pediculosis

This means infestation by lice. There are 3 types (Fig. 45):

1. *Pediculus capitis*—head louse.
2. *Pediculus corporis*—body louse.
3. *Phthirus pubis*—crab louse.

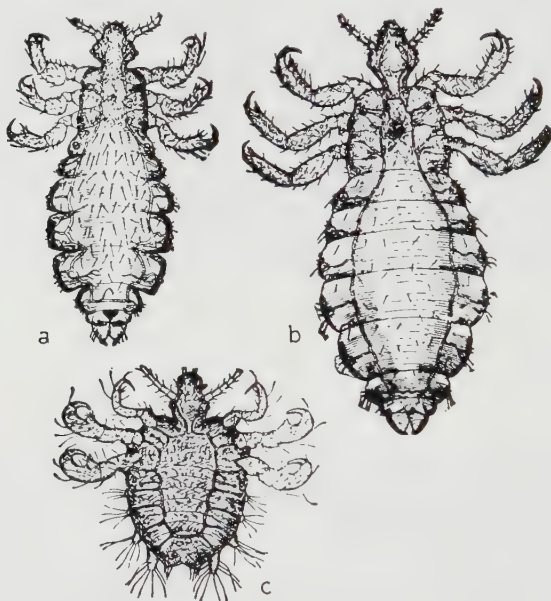
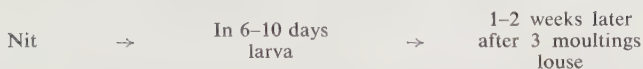


FIG. 45. Pediculi: dorsal surfaces of females. (a) *P. capitis*, (b) *P. corporis*, (c) *P. pubis*.

PARASITOLOGY

Lice are oval and grey, about 2–4 mm long, wingless, and have 6 legs. Pubic lice are the smallest, and body lice are the largest. The female lays several hundred eggs, called nits, each one being glued on to a hair. A larva is hatched out in 6–10 days, and becomes a fully-grown louse in 1 or 2 weeks.



They live on blood, which they suck from the skin, and only attack live humans. Each louse rarely leaves its own territory, except in severe infestations.

Pediculosis Capitis

This is found chiefly in women and girls, especially at the back and sides of the scalp. As a result of scratching, impetigo occurs, and enlargement of the occipital glands. The eyelashes may also be involved. The translucent nits can be seen glued to the hair (Fig. 46), and are difficult to remove. A watchful eye may see a louse moving on the scalp.



FIG. 46. Nit or ovum glued to a hair by a cylindrical sheath of chitin.

The diagnosis must be firmly made before informing the patient. Infestation is considered by some to be a terrible disgrace, and if the patient is wrongly informed the physician's reputation will suffer disproportionately.

TREATMENT

2 per cent DDT emulsion is rubbed into the hair once, or 10 per cent dusting powder shaken into it. DDT remains to some extent even after washing, and lice are killed by traces of it. When DDT is unavailable 25 per cent benzyl benzoate may be used. The hair need never be cut. Contacts must not be excluded from treatment.

Pediculosis Corporis

This louse lives in clothes, and only leaves them to have a meal off the skin. The eggs are laid in the underclothing. Scratch marks are the only lesion of this infestation. Body lice are the cause of typhus, trench fever, and relapsing fever.

TREATMENT

is by giving the patient a hot bath, and following this with the application of 10 per cent DDT powder, not forgetting the underclothes.

Pediculosis Pubis

This may be contracted during sexual intercourse, or from bedding. It is commonly found in the pubic area, but also in the axillae, and on the eyelashes and chest. Itching may be severe, and the louse should be looked for in cases of pruritus ani and vulvae.

TREATMENT

is by an application of 2 per cent DDT cream, or 25 per cent benzyl benzoate emulsion. Contacts must also be treated.

Fleas

They are brown, wingless, flat insects, with 3 pairs of legs, and have the power to jump huge distances in relation to their size. Their bite (to which many people are relatively immune) produces a haemorrhagic spot surrounded by an itchy wheal. They may also be the cause of papular urticaria.

TREATMENT

is by spraying carpets, cracks in floors, domestic animals and their baskets with 10 per cent DDT powder. To catch a flea is an art; a piece of moist soap and a white surface like a bath or a sheet are required for it to jump on, as it is inclined to be attracted to such a colour. A steroid lotion is a good anti-pruritic.

Bedbug

It is yellowish-brown, oval, with 3 pairs of legs, and is twice as big as a louse. It is said by specialists to have a nasty smell. It lives in cracks in furniture, and can do so without food for a year. They may be the cause of papular urticaria.

Bites are grouped in twos or threes, especially round the ankles and buttocks.

Crevices in furniture should be sprayed with DDT.

Bees, Wasps, Ants

Chronic urticaria can result from the sting of these insects, as well as generalized formication and granulomata.

Bee poison is acid and ammonia or sodium bicarbonate should be applied, after extraction of the sting.

Wasp poison is alkaline, or neutral, and should be treated with lemon juice or vinegar.

Insect bites occasionally produce bullous lesions.

Antihistamines by mouth are useful for reducing pruritus and oedema.

CHAPTER 14

Disturbances of Pigmentation

Pigment may be increased in the skin, or, in some conditions, diminished; however, hyper-pigmentation is more common than hypo-pigmentation. There are a great many conditions which come into both categories, and only the common ones will be dealt with here.

The principal causes for hyper-pigmentation are:

- (1) Increase of melanin, which may be local or general, and more rarely:
- (2) Deposits of foreign pigments.
- (3) Deposits of heavy metals, e.g. arsenic.
- (4) Deposits of haemoglobin derivatives, as in haemosiderosis.

The common conditions characterized by hyper-pigmentation are:

Local

Freckles

Pigmented naevi

Chloasma

General

Thyrotoxicosis

Addison's disease

Chronic hepatic insufficiency

Scleroderma

Freckles

are caused by sunlight. They are common in blonde individuals and tend to fade in winter. There is no effective therapy, apart from the prophylactic avoidance of sunlight.

Chloasma

describes brownish patches on the face of the pregnant woman, due to the melanocyte-stimulating hormone of the pituitary gland. They disappear at the end of pregnancy.

Pigmented naevi

are dealt with in chapter 20.

Common conditions characterized by hypo-pigmentation are:

Local

Vitiligo

General

Vitiligo

Albinism

Albinism

This is a congenital absence of melanin involving the hair, eyes, and skin.

It is commoner in males, and there is often a familial or hereditary history.

The skin is a delicate pink, the hair a silky white, the eyes are pink, and the person's sensitivity to light is noticeable: he blinks his eyelids and stands with cast-down eyes. Keratoses and epitheliomata are liable to occur later in life, as a reaction to the activity of the sun.

No treatment is effective, but the skin and eyes must be shaded from sunlight.

Vitiligo or Leucoderma

A condition in which pigment disappears from the skin in patches, so that they become white (Fig. 47).

CAUSE

This is unknown, although there is sometimes a familial history. Dark people are commonly affected, and both sexes at any age.

CLINICAL FEATURES

The onset is sudden. The patches assume various sizes and shapes and are commonest on the face and neck, hands and wrists, abdomen and thighs. Hair may also become white. All these patches are very sensitive to sunlight.

COURSE

The condition may remain static for months or years, and only occasionally clears spontaneously.



FIG. 47. Vitiligo (leucoderma) of the leg.

DIAGNOSIS

In widespread cases it is easy to confuse the vitiliginous condition, with one of hyper-pigmentation. It may seem that the individual has a white skin peppered with dark patches which, in contrast with the white, are very dark indeed. The true state of affairs can be made

clear by examining the edge of the white areas. If it is convex, the condition is one of vitiligo.

TREATMENT

None is curative, but various cosmetic creams may be applied to disguise exposed areas. Innoxia Ltd, of London, make an effective cream, named Keromask.

CHAPTER 15

Diseases due to Metabolic or Hormonal Disorders

In these conditions, the skin lesions play a very minor role as regards the health of the patient, but their recognition, in some cases, is a major clue to the underlying diagnosis. The condition most noticeably absent from this chapter is acne vulgaris, which is dealt with on page 187.

METABOLIC DISORDERS

Xanthomata

These are yellowish or pinkish—yellowish papules, or nodules (Plate 22), containing cholesterol fat. There are two varieties.

(1) Xanthoma planum

in which yellow papules or plaques form in the skin of the eyelids, generally of middle-aged women. The blood cholesterol is raised in over two-thirds of all cases.

TREATMENT

consists of excision, or the very careful application of tri-chlor-acetic acid, which results in a scab, which disintegrates within 2 or 3 weeks. In many cases, no treatment is required.

(2) Xanthoma tuberosum

which, as might be gathered, consists of larger, nodular lesions. They appear on the extensor surfaces. The mucous membranes may also be involved. The blood cholesterol is always raised. Large nodules can be excised.

Vitamin Deficiencies

Vitamin A

This is characterized by dryness and roughness of the skin, usually of the fronts of the thighs and the back of the forearms. The hands and feet usually remain unaffected.

Vitamin D

This causes no skin lesions.

Vitamin E

There is no concrete evidence that this is responsible for skin lesions.

Vitamin B₁

This causes no skin lesions, unless combined with vitamin B₂ deficiency, when pellagra ensues.

Pellagra

This is characterized by symmetrical redness and scaliness of areas exposed to light, namely, the wrists, ankles, face and neck. Weeks or months later, peeling occurs and is followed by pigmentation.

The pellagrinous nose—redness and scaling of the bridge—is characteristic.

Pellagra is recognized by the 3 D's of dermatitis, diarrhoea and dementia.

Other signs of this deficiency are a sore tongue, inflamed lips, anorexia, weight loss and insomnia.

TREATMENT

is by giving nicotinamide 500 mg daily by mouth, and 50 mg daily intra-muscularly, apart from bed-rest, and a diet rich in the vitamin B complex.

Vitamin B₂ (Riboflavin)

Skin lesions are characterized by redness and fissuring of the angles of the mouth (angular cheilitis). In severe cases, the tongue becomes magenta coloured.

Vitamin C (Ascorbic Acid)

The principal signs of scurvy are: (1) redness and bleeding of the gums; (2) petechial haemorrhages of the skin, especially in the tourniquet test; (3) easy bruising and slow healing of wounds.

HORMONAL DISORDERS

Acromegaly

There may be many skin changes, and these include thickening and furrowing of the skin of the face, with seborrhoea and hirsuties. The nose becomes broad, and the ears, lower lip and tongue markedly thicker. The nails become broad, thickened, and sometimes spoon-shaped.

Cushing's syndrome

Many notable skin changes may accompany this disorder, which is due to a basophil adenoma of the pituitary, or due to hyperplasia or a tumour of the adrenal cortex.

The skin may be hirsute, dusky and plethoric, accompanied by purpura and ecchymoses, and remarkable striae distensae.

All these signs may arise too as a result of steroid administration.

Myxoedema

Hypothyroidism

In this condition, the skin, hair and nails are affected. The skin is dry, uncomfortably rough, swollen and waxy, this colour being due to carotenaemia. Although the skin is swollen, there is no pitting oedema. The hair of the scalp is dry and thin, and has none of its normal lustre. Examination of the hair of the eyebrows will reveal a definite loss of their outer two-thirds, and pubic and axillary hair will be absent or very sparse. The nails are brittle and ridged.

A *localized* form of the above condition may occur on the fronts of the legs, and is called pre-tibial myxoedema. It may or may not be associated with thyroid insufficiency.

Diabetes Mellitus

This condition may be accompanied by boils or carbuncles, pruritus, leg ulcers, or xanthomata.

Boils and pruritus are such common conditions that, in a busy clinic when all urines cannot be tested, special attention should be paid to urine examination when there is a history of boils or pruritus, even though the test has been made elsewhere! The number of cases in which glycosuria is present is very small, so that sometimes one

despairs of finding sugar in the urine. But when one is least expecting it, a case will arise, rewarding perseverance.

Addison's Disease

The pigmentation in this disease is light to dark brown, and affects exposed surfaces, except the palms and creases.

Pregnancy and skin lesions

The incidence of eruptions during pregnancy is less than 1 per cent. They usually appear after the fifth month, and disappear early in the post-natal period. Various erythematous eruptions may occur, and pruritus or urticaria.

CHAPTER 16

Diseases due to Vascular Disorders

The commonest skin disorders under this heading are those due to stasis in the vessels, and comprise such conditions as varicose dermatitis, thrombo-phlebitis of the leg, and cardiac failure producing back-pressure. Purpuric eruptions also come under this heading.

Varicose Dermatitis and Ulcer

The skin conditions produced by varicose veins are dermatitis and ulceration and they are, unfortunately, very common in the practice of skin diseases.

The cause of the eruption is nearly always a former thrombo-phlebitis, which in a great many cases the patient cannot recall, either because it occurred a few years before the appearance of the skin lesions, or because the discomfort was so minimal that it passed unnoticed. Middle-aged women outnumber men by about 4 to 1. Overweight and heredity may play minor roles in the causation.

CLINICAL FEATURES

The onset is insidious with moderate oedema of the inner aspects of the ankles. This tends to wax and wane, depending on the amount of standing the patient has to do. (Walking is far less harmful as it constantly maintains a venous flow.) Poorly-defined areas of redness then develop, and gradually dry or moist scaly lesions appear. These become itchy, and owing to scratching or minor trauma, an ulcer develops. The ulcers have soft irregular edges, become infected, and contain a most offensive smell. They vary in size from a halfpenny to involvement of large areas of the leg (Fig. 48), and can be very painful. Both legs may be affected.

As the disease progresses, there is always a danger of eczematization of the skin, and an eczematous eruption occurring at first around the ulcer may then spread widely over the body.



FIG. 48. Varicose ulcers: 10 years' recurrent ulceration (Skin Department, London Hospital).

DIAGNOSIS

When dermatitis is present, localized neurodermatitis must be excluded. This is never associated with oedema of the ankles, and is extremely itchy.

When an ulcer develops, hypertension and arteriosclerosis must be considered, but the ulcers are small and punched out, and usually extremely painful. Malignancy gummata and erythema induratum should be considered in some cases, but there is no evidence of venous stasis. Less common causes of leg ulcers are, for example, chilblains, ulcerative colitis, and rheumatoid arthritis.

TREATMENT

For thrombophlebitis

firm bandaging, and anti-coagulants if necessary.

For dermatitis

a bland ointment or cream containing calamine or titanium dioxide (Siccolam) or a steroid cream are often effective. Nothing must be applied which could possibly cause sensitivity of the skin, which is, in this condition, in a very delicate state. Antibiotic ointments should only be given after careful thought. If the rash is very severe, a short term of bed-rest may be necessary. When the rash has cleared, supportive treatment for the leg in the form of elastic stockings or bandages must be instituted indefinitely. A surgeon's opinion should be sought concerning the advisability of operative treatment.

For ulcers

the aim is to avoid anything encouraging venous stasis, and the accumulation of oedema, and to improve the blood supply.

Bed-rest should be avoided unless there is gross infection of the ulcer and surrounding oedema. Even then, it should only be employed to bring the condition initially under control. Then the foot of the bed should be raised, and a cradle introduced under the bed-clothes to take their weight off the leg. Long periods of bed-rest are definitely harmful.

Ambulant treatment is best for ulcers, and this is made possible by bandaging. The following two methods are effective:

1. Occlusive paste medicated bandages. A zinc or calamine bandage impregnated with iodochlorhydroxyquinoline (Quina-band) is applied to the leg from the knee to the foot, and changed at weekly intervals.
2. Crepe bandaging. A polyurethane foam pad is placed over the ulcer and kept in position by a length of tubular gauze.

Compression is then obtained with a cotton crepe or poroplast bandage. This outer bandage may be removed at night-time, and reapplied each morning.

Finally, physiotherapy in the form of massage and exercises is very helpful in reducing oedema and induration.

PROGNOSIS

The future health of the leg depends on the patient's recognition of the importance of avoiding trauma of any kind, of avoiding long periods of standing, and the wearing of a supportive elastic bandage, when the slightest oedema is present.

Purpura

This term is used to describe the appearance of purple-red spots in the skin or mucous membranes, due to haemorrhage from capillaries. It is not a disease in itself, but a sign of an underlying condition. The causes are innumerable, and sometimes difficult to discover.

The causes may be grouped under two headings: (A) Capillary wall defects; (B) Blood defects.

(A) CAPILLARY WALL DEFECTS

This may be a generalized or a localized condition. If the former, the tourniquet test will be positive. If the latter, it will be negative. In both cases, the platelet count should be normal, i.e., 200,000–500,000.

The following are common causes of this variety of purpura:

1. Simple purpura.
2. Senile purpura.
3. Venous-stasis purpura.
4. Drugs; also responsible for purpura associated with blood defects.
5. Avitaminosis; e.g. scurvy.
6. Systemic diseases; also responsible for purpura associated with blood defects.
7. Henoch-Schoenlein syndrome.

1. SIMPLE PURPURA

This mild form of purpura occurs in children affecting the legs, forearms and the mucous membranes of the mouth. It clears up in about a month, although it is likely to recur over the years. Slight constitutional symptoms may or may not be present.

2. SENILE PURPURA

This is similar to the childhood variety, but is due to degeneration of the tissue supporting the capillaries.

3. VENOUS STASIS PURPURA

This variety occurs around the ankles and lower parts of the legs due to varicose veins, and also in fat men in the absence of varicose veins. It is accompanied by progressive pigmentation and the deposition of haemosiderin, so that a cayenne-pepper appearance develops.

4. DRUGS

Many drugs can produce a purpuric reaction. Among a few commonly responsible, the following should be noted: sulphonamides, chloramphenicol, gold, and carbromal, a drug used to induce sleep.

5. SYSTEMIC DISEASES

Again only a few will be named. These are: subacute bacterial endocarditis, scarlet fever, measles, meningitis, typhoid fever, diabetes, hypertension and chronic nephritis.

6. HENoch-SCHOENLEIN SYNDROME

This type occurs in children and rarely in adults, and is characterized by a special sequence of lesions, as well as gastro-intestinal and arthritic symptoms.

CLINICAL FEATURES

These are commonly preceded by an upper respiratory infection occurring one or two weeks before other signs.

The earliest lesion is a dark red purpuric papule, which becomes successively vesicular or bullous and then ulcerates. Sites commonly affected are the buttocks and the extensor aspects of the limbs, the distribution being symmetrical.

The arthritic pains most commonly affect the knee and ankle joints.

The gastrointestinal symptoms are in the form of epigastric pain, and sometimes haematemesis, often initiating appendicitis or intussusception.

TREATMENT

Bed-rest, which clears up the attack in a week or two. In severe cases, steroids have been used with success.

(B) BLOOD DEFECTS

The common defect is thrombocytopenia. Defects in clotting, and plasma protein abnormalities are rare.

When purpura is present, a platelet count should always be made.

The common conditions producing thrombocytopenic purpura are as follows:

1. Idiopathic, or primary thrombocytopenic purpura.
2. Drug purpura; also a cause of the non-thrombocytopenic variety.
3. Systemic diseases: also responsible for the non-thrombocytopenic variety.

1. IDIOPATHIC THROMBOCYTOPENIC PURPURA

There are two forms: (i) acute, and self-limiting, occurring in children; (ii) chronic, being associated with splenomegaly, and found in adults.

For the chronic form, the following treatments should be considered: steroid therapy, and splenectomy.

2. DRUG PURPURA

See under purpura due to capillary defects.

3. SYSTEMIC DISEASES

Here other more grave conditions should be considered, such as leukaemia, acute or chronic, aplastic anaemia, splenic disorders such as Kala-azar or malaria, or generalized carcinomatosis.

TREATMENT

The treatment of all purpuric eruptions depends on the cause, and when the condition is systemic a general physician should be consulted.

CHAPTER 17

Systemic Diseases of Unknown Cause

LUPUS ERYTHEMATOSUS

This condition appears in two distinct forms:

1. Localized, or chronic.
2. Generalized, or acute.

Chronic

The *chronic type* is characterized by inflammatory, scaly, round papular lesions, usually distributed symmetrically on the face (Plates 23, 24). It commonly remains localized to the skin, but may occasionally become systemic and fatal.

CAUSE

This is unknown, although the disorder appears to be one of the collagen tissue, and an allergic auto-immune reaction (see acute L.E.).

PREDISPOSING FACTORS ARE

Sex: women are more affected than men.

Age: the usual incidence is between 30 and 50.

Climate: those living in northern zones are more prone than others.

PATHOLOGY

The classical changes in this disease are: (1) hyperkeratosis, which notably plugs the follicles (Fig. 49), and which is also a diagnostic clinical sign; (2) alternating acanthosis and atrophy of the prickle-cell layer; (3) liquefaction degeneration of the basal layer, so that this layer is separated by a cleft from the dermis; (4) a peri-vascular

lymphocytic infiltrate seen chiefly around the appendages of the dermis. Other less typical changes may or may not be present.



FIG. 49. Chronic lupus erythematosus showing the plugging of follicles by scales, so-called follicular hyperkeratosis.

CLINICAL FEATURES

The onset is insidious. The lesions are papular, usually about pea-size, and enlarging very slowly to half-crown size or larger. They are normally round or oval, and very close examination of the scaly surface will reveal that the scales in many areas appear as dots; these dots indicate where the follicle has been plugged by a scale, and when scales are removed and the undersurface is inspected they clearly appear as tiny spicules projecting from the scaly mass. No other scaly condition produces this phenomenon.

Sites commonly affected are the face, particularly the cheeks and nose (the so-called butterfly area of the face, the cheeks being the imaginary wings of the butterfly, Fig. 50), the ears and the scalp. The mucous membranes of the lips and mouth may be attacked and show sharply defined, slightly raised whitish patches, with red

borders. When the lips themselves are involved, they are covered with very closely-bound scales.



FIG. 50. Discoid lupus erythematosus: involvement of the so-called butterfly area of the face (Department of Dermatology, Addenbrooke's Hospital).

Investigations

Blood count, urine, and sedimentation rate should be checked, as well as an L.E. cell preparation, and all these should be negative in chronic lupus erythematosus (see acute L.E.).

COURSE

The lesions heal with flat and slightly depressed scars. Relapses are common, and may continue for many years. But above all, a watchful eye must be kept on all active cases of chronic lupus erythematosus. A small percentage of them may develop into the dreaded systemic type, described below. This is refuted by some authors, who consider the acute and chronic types to be different entities. There is no unanimity about this.

DIAGNOSIS

This is made by the appearance of scaly discoid lesions (Fig. 51), with the unique plugging of the follicles. The condition must be distinguished from (1) *lupus vulgaris*, which tends to ulcerate the skin, and in which there is no follicular plugging; (2) *eczema*, which is itchy.



FIG. 51. Discoid lupus erythematosus. Diffuse facial distribution of lesions (Dr E. Waddington).

and whose evolution is more rapid; (3) *psoriasis*, which has loosely-adherent silvery scales, usually with evidence of the condition elsewhere; (4) *seborrhoeic dermatitis*, which shows greasy loose scales; (5) when appearing on the hands, *chilblains* must be excluded.

TREATMENT

Patients must avoid sunlight, and when out-of-doors wear wide-brimmed hats. Antibiotics must be avoided, as they may disseminate the disease.

The following treatments may be used:

Steroids applied as creams or ointments.

Steroids by injection into the lesion.

Steroids by mouth.

Chloroquine sulphate (Nivaquine) or

Hydroxy-chloroquine (Plaquenil) tablets.

Betamethasone valerate (Betnovate) and fluocinolone acetonide (Synalar) creams rubbed in twice daily suppress most lesions. For deeply infiltrated lesions, intralesional injections of triamcinolone can be given. Chloroquine (Nivaquine) 200 mg tablets 2 or 3 times a day may be given as a last resort, providing no side-effects occur (see p. 93), and even so, used only during spring and summer months under constant supervision.

Acute

The *acute disseminated type* of lupus erythematosus (systemic L.E.) is a progressive and grave disease. It affects every organ in the body, and in only half the cases is there skin involvement.

CAUSE

This is unknown, although there is substantial evidence that it is due to an allergic auto-immune reaction, as there is antibody formation, and the production of auto-antibodies, e.g. the L.E. phenomenon, and Coombs' test.

The patient stricken with systemic L.E. is particularly vulnerable to many reactions which may act as a trigger mechanism. The effect of sunlight on a skin lesion may result in the development of an

acute exacerbation. Drug reactions to sulphonamides and penicillin may also produce the same effect.

Another pointer to the cause may lie in the fact that collagenous tissue is the target area, degenerative changes being found in the mesenchymal tissues of all organs attacked.

The L.E. Phenomenon

This phenomenon is seen in blood smears, as a result of a special examination of the patient's plasma or serum, when mixed with normal heparinized marrow, or whole blood. The L.E. cell may then be seen. It is a leucocyte, engulfing a round, smoky, basophilic mass of nucleoprotein.

The L.E. phenomenon is always positive at some time in systemic L.E., and only in chronic L.E. when a transition from the chronic to the acute is taking place.

PATHOLOGY

Skin changes when present are relatively similar to those of the chronic variety.

Visceral changes commonly found are: (1) verrucous endocarditis (Libman-Sacks syndrome); (2) renal lesions producing a nephrotic syndrome; (3) pleurisy or pericarditis. A vasculitis of the C.N.S. may also occur, and the skeletal muscles may be affected.

CLINICAL FEATURES

Languor, weakness, anoxia, loss of weight, low-grade pyrexia, chest pains, vague joint symptoms.

Skin lesions are present in only 50 per cent of cases (Plate 26). Other clinical signs are legion, as any organ may be attacked.

Laboratory investigations will reveal albuminuria, haematuria, leucopenia, anaemia, thrombocytopenia and a very high sedimentation rate.

DIAGNOSIS

If classical skin lesions are present it is straightforward. If not, it is based on the laboratory findings and clinical signs. Rheumatic fever and bacterial endocarditis must be excluded.

TREATMENT

Sunlight and offensive drugs must be avoided.

Bed-rest, good nursing and feeding are essential. Steroids must be given, and at first in high doses; e.g. prednisolone 300–400 mg daily. Reduction of the dose must be gentle and slow. Anti-malarials, e.g. chloroquine, also have a place in treatment.

This is but an outline of the treatment of one of the most challenging diseases in medicine.

PROGNOSIS

This is always grave, and depends on many factors such as the extent of the disease and its response to steroids.

Sarcoidosis

This is a chronic systemic disease, which produces papules, nodules or plaques in the skin.

The pathological reaction is a reticulo-endothelial one, and the disease prefers organs containing a large amount of reticulo-endothelial tissue.

The term sarcoid is derived from the Greek word *sarx*, meaning flesh. Sarcoid is a misleading term, as it suggests a relationship to sarcoma, which does not exist. Like many other misnomers in medical terminology, it is too deeply embedded to be eradicated.

The condition is commonest in females, and between the ages of 20 and 40.

CAUSE

This is unknown. The disorder bears a faint resemblance to tuberculosis, clinically, but the relationship to it is very doubtful. A viral cause has been cited but never proved.

PATHOLOGY

The most characteristic feature is the presence of well-defined clumps, resembling nests, of epithelioid cells. Once seen, they are unmistakable. Their position in the skin varies according to the type of skin lesion present. This epithelioid infiltration is the same whether it involves the lungs, bones, viscera or skin. Caseation is rare, giant cells are occasionally present, and tubercle bacilli never.

CLINICAL FEATURES

Skin changes occur in nearly 50 per cent of cases of sarcoidosis. The commonest cutaneous indication is erythema nodosum (see p. 83). But specific skin changes also occur.

The onset is gradual. The lesions are papules, nodules or plaques. They may be any shape, and are quite well defined (Plate 26). They are reddish-brown, and vary in number from one to a hundred or more. The surface of the skin is usually unchanged, and the granulomatous infiltrate can be sensed by palpation. This varies in degree, depending on whether the infiltrate is dermal or subcutaneous. The commonest sites are the face, neck, and back of the arms.

Other aids

to diagnosis are by means of diascopy (see p. 116), when the lesion shows a brownish colour similar to lupus vulgaris.

The blood may show a marked increase in proteins, in particular a hypergammaglobulinaemia.

The Kveim test involves the injection of a ground-up suspension of sarcoid tissue, which produces a sarcoid reaction if the disease is present.

COURSE

The lesions usually fade without leaving a mark, although occasionally atrophy and scarring occur.

Other signs

which may occur are very varied, and consist of (1) lymphadenopathy, in 80 per cent of cases, usually involving the cervical, axillary, or inguinal groups; (2) lung changes in 75 per cent of cases; (3) bone involvement, presenting as a non-ulcerative dactylitis of the fingers and toes; and (4) granular uveitis, which may lead to blindness.

DIAGNOSIS

This is made by the Kveim test, and the histology. Like other conditions which show a multiplicity of non-specific lesions, it is a diagnosis which must be consciously brought to mind.

Sarcoidosis must be distinguished from other granulomatous conditions such as *tuberculosis*, *syphilis* and *leprosy*. *Reticuloses* and *drug eruptions* must also be considered.

TREATMENT

When the condition is localized to the skin, none is indicated, as the lesions ultimately resolve without it, but should it be apparent that the disease is systemic, steroids must be considered.

PROGNOSIS

Cutaneous lesions tend to recur.

SCLERODERMA

This is a disorder characterized by induration of the skin. It exists in two forms: (1) localized; (2) generalized. The cause is unknown.

PATHOLOGY

The chief changes are fibrosis and sclerosis of the collagen fibres, and sclerotic and obliterative changes in the vessels. In later stages, there is universal atrophy of the appendages in the dermis, and elastic tissue becomes fragmented.

When the condition is generalized, grosser changes occur, such as sclerosis and atrophy of the skeletal muscle bundles. The oesophageal, intestinal and cardiac musculature is also often involved, and is clearly reflected in the clinical signs.

Localized Scleroderma

This is also known as morphoea, and consists of localized patches of hardened white atrophic skin.

CLINICAL FEATURES

The onset is gradual. The lesion may be pea to palm size, varying in its rate of growth. The shape may be oval, round or linear, and in recent lesions is edged with a definite pale violet hem. Later it disappears. The surface is hard and smooth, and the lesion is difficult to lift. There may be one or several lesions, and any site can be involved.



PLATE 24. Lupus erythematosus. Chronic variety, showing well-demarcated scaly lesions on the face and left eyebrow, simulating seborrheic dermatitis (Skin Department, London Hospital).

PLATE 25. Lupus erythematosus. Diffuse erythema of the face, in a case with systemic involvement (Dr G. A. Hodgson).





PLATE 26 Sarcoid, in this case simulating rosacea (Dr Martin Beare).



PLATE 27. Seborrheic warts.

Other varieties

(1) *Guttate form* consisting of very white round drop-like atrophic macules. The lesions may become larger by coalescence. (2) *Coup de sabre*, which presents itself as a wide sabre-shaped band extending from the forehead towards the vertex, lying close to the median line. It may be associated with epilepsy and atrophy of one side of the face.

COURSE

The lesions remain indefinitely, or slowly resolve. Nearly 5 per cent imperceptibly alter into the grave systemic form of scleroderma, particularly if there are many lesions.

Systemic or Progressive Scleroderma

This is a rare, very serious, chronic condition, characterized by the progressive induration and atrophy of connective tissue all over the body. It commonly affects women between 30 and 50, although no age or sex is exempt.

CLINICAL FEATURES

The onset is insidious. The skin changes are usually preceded by signs of Raynaud's phenomenon. The skin becomes hard and stiff, and movements become severely restricted. The face, hands, and forearms are first attacked, and soon the face is contracted and immobile, the lips thin and pinched and the chin puckered. Mastication becomes very difficult, and the hands cannot be closed into a fist, and later they are useless. Trophic ulcerations and gangrene are common terminal sequelae. Systemic changes follow, such as severe dysphagia, dyspnoea, and cardiac insufficiency.

COURSE

The spread varies in rate, but is relentless.

DIAGNOSIS

This is made by the characteristic contracted facies, and the restricted movements.

Raynaud's disease is characterized by paroxysmal attacks of ischaemia, with numbness and paleness. *Syringomyelia* presents dissociated areas of anaesthesia.

TREATMENT

Steroids often are beneficial in the early stages of the disease.

Physiotherapy and exercises are essential in an effort to postpone contractions. Cold is to be avoided, and patients should, if possible, live in a warm climate, but there is no treatment which can guarantee permanent relief.

Dermatomyositis

This is an acute, subacute, or chronic disorder characterized by dermatitis of various types, oedema, muscular pains, non-suppurative inflammation, and degeneration of the muscles.

The cause is unknown, both sexes are equally affected and no age is exempt but it is commonest in the fifth decade. It is an uncommon condition.

CLINICAL FEATURES

The onset is insidious or sudden, and may follow an infection. Puffiness of the face develops and, notably, of the eyelids and malar area. The puffiness recedes leaving a reticulated telangiectatic erythema, and an appearance of atrophy. Muscular pain and tenderness vary, and any muscle may be affected.

Carcinoma of various organs is not uncommon, and is the usual cause of death.

TREATMENT

Rest, but not absolute bed-rest, and high calorie diets. Steroids may suppress the disease. Physiotherapy may help prevent contractures.

PROGNOSIS

The course is unpredictable. Some patients die within a few weeks to a few years of onset. Others recover, or become chronic cases indefinitely.

CHAPTER 18

Diseases of the Appendages

Acne Vulgaris

This is a chronic inflammatory disease, characterized by comedones (blackheads), papules, pustules, and sometimes cysts, involving the sebaceous glands. Owing to thickening, or hyperkeratosis, of the horny layer at the orifices of the hair-follicles, the sebum becomes plugged, giving rise to the lesions described.

CAUSE

The exact cause is unknown.

Sex

The condition is slightly more common in girls.

Age

Any time after puberty until the age of 30.

Heredity

There is a greater tendency for acne to develop when there is a familial history of the disease.

Season and Climate

Acne is more common in winter, and in temperate zones.

Hormones

The age of onset of acne coincides with a momentous hormonal revolution. This factor is largely responsible for the increase in size and activity of the sebaceous glands, and for the thickening of the horny layer, already mentioned, which results in comedones, or

blackheads, so-called because the plug of keratin becomes black or dark brown as a result of oxidation. Some hormones secreted at this age are androgenic substances in the male, and progesteronic in the female. What role exactly they play in the production of acne is unknown; in some cases it may be significant, in others it may not. On one hand, an excessive supply of male sex hormone may be concerned, although no significant increase in androgen excretion has been found, whilst on the other, a reduction of oestrogenic substances, which has been found, may be important.

PATHOLOGY

Examination of a comedo shows that it is composed of sebum, and horny and uncornified cells, lying in a pilo-sebaceous follicle. It is surrounded by an inflammatory infiltrate of varying intensity, which accounts for the redness around the lesion. When pustular lesions are present, pus cells are found, sometimes with the formation of small abscesses, and disintegration of the follicle.

CLINICAL FEATURES

There are no symptoms; only those of embarrassment which may be latent unless the patient is questioned on the point. This is the result of bad medical or parental advice, which consists mainly of stating that acne is a normal event in the process of growing up, and that the spots will disappear with time. Both statements are to a certain extent true, but they do not invalidate the necessity for energetic treatment which will hasten recovery.

The onset is gradual. The lesions consist of comedones, papules and pustules, and occasionally cysts. They are found on the face (Fig. 52), chest and back, and back of the neck. In number they vary from a few to hundreds; the skin of the acne patient is often greasy, and the complexion, brunette. Dandruff is commonly present in varying degrees.

Other varieties

Excoriated acne occurs as a result of neurotic and intensive picking of lesions, and is usually seen in females.

Infantile acne occurs in babies, when food from the bottle or the breast includes too much cream.



FIG. 52. Acne vulgaris.

Acne keloid is a chronic staphylococcal infection of the hair follicles of the back of the neck of men, which result in the formation of unsightly hypertrophic scars. Although somewhat dissimilar, it bears certain resemblances to true acne.

Acne conglobata is a rare variant of acne, characterized by the formation of abscesses, sinuses, and scars.

Occupational acne is not a variety of *acne vulgaris*, but produces similar lesions, as a result of the person working in contact with oils, waxes, tars, or chlornaphthalenes.

DIAGNOSIS

This is usually quite simple, and should only be made when comedones are present. *Rosacea* may imitate *acne vulgaris* to a certain extent, but there are no comedones, the condition is limited to the nose, cheeks, chin, and forehead, and it occurs after the age of 30. *Iodide and bromide eruptions* produce pustular lesions, and can be excluded by careful history-taking. The same care applies to the exclusion of *occupational acne*.

TREATMENT

An optimistic approach must be made with whatever form of treatment is employed. Plenty of rest, exercise and sleep should be emphasized, advice which young people often find difficult to follow. Frequent washing of the face, i.e. at least four times a day, should be encouraged with soap and warm water. When the eating of chocolates and fatty foods appears to produce more lesions, these foods must be avoided. Squeezing and picking of the lesions must be avoided. Contrary to the belief of some, marriage does not cure acne.

External

Sulphur is the best local application, as it causes mild peeling of the skin. It is best used in a lotion, as a cream is redundant to an already greasy skin. Sulphurated potash and zinc lotion B.N.F. is the most universally used sulphur-containing lotion. If a cream has to be used on account of excessive dryness of the skin, 5 per cent sulphur in equal parts of water and an emulsifying cream can be prescribed. Pastes, although they are effective peeling agents, are not advised because they are so unsightly.

Brasivol ointment, which consists of finely-ground aluminium silicate in an emulsifying base, is useful in some cases as a mild abrasive, as is the Comédome abrasive stick containing sulphur and hexachlorophene. A more satisfactory method of gently and temporarily exfoliating the skin is by an ultra-violet radiation (u.v.r.).

Exposures producing a 2nd degree erythema may be given twice a week. U.V.R. is particularly helpful in brunettes, but on blonde skins may produce ill-timed discomfort and therefore should be used with caution.

CO₂ slush, which consists of ground CO₂ snow mixed with acetone, can be swabbed over the affected areas, and this also produces a peeling effect.

X-rays in appropriate doses cause reduction in the size and activity of the sebaceous gland, and therefore may be employed in carefully selected cases of those over the age of 17. Many dermatologists are opposed to this form of therapy.

X-rays do not remove blackheads. This is best done by means of a comedo expressor. This is a spoon-handled object, with a hole in it. When pressed on the spot, the comedo exudes through the orifice. Dermabrasion is a little-used method of planing skin which is scarred; it is performed with a revolving burr, resembling a dentist's drill.

Internal

Broad-spectrum antibiotics of the tetracycline group are the most useful form of this treatment and should be tried when the lesions are predominantly pustular, or cystic. Acne vulgaris is not an infective condition, and the pustules themselves are sterile, but in many cases antibiotics such as Achromycin, or Terramycin in a dosage of 250 mg three times daily for a week, and subsequently twice daily, is often the only form of treatment which maintains a reasonably presentable appearance. Why this is so is a mystery. Penicillin and sulphonamides are rarely useful.

Hormones can be used in cases where there seems to be a particular relationship of the appearance of lesions to the menstrual cycle. Ethinyl oestradiol 0.1 g t.d.s. for 10 days after the last day of the period may be helpful.

Thyroid extract in small doses is sometimes helpful. Vaccines are useless.

PROGNOSIS

All patients can be improved. 90 per cent should be cured except for the appearance of an occasional lesion, but this depends on the

confidence of the patient in the treatment, and the doctor's ability to impart it. Furthermore, the patient should accept the fact that a cure will take several months.

Seborrhoea

This term implies that there is an increased flow of sebum.

It is an inborn and common characteristic of many people, and produces excessive oiliness of the skin, particularly of the nose and central areas of the face, whose follicles tend to be patulous.

The hair becomes greasy, and frequent shampooing allays symptoms for only a day or two. The condition usually occurs and lasts as a post-pubertal state for a few years, but occasionally persists indefinitely.

Treatment consists of frequent washing with soap and water, and shampoos.

Sebaceous Cysts

These are smooth, round, globular, cutaneous or subcutaneous tumours which arise from the sebaceous glands, and are found on the face, neck, scalp, back and genitalia.

CAUSE

Unknown. Occasionally hereditary.

PATHOLOGY

The cyst contains cheesy, smelly material, consisting of masses of degenerating and disintegrating epithelial cells. It is encapsulated by fibrous connective tissue.

CLINICAL FEATURES

The size of the tumour varies from a pea to an orange or larger. The surface is smooth and shiny. To palpation they are soft and doughy, or firm. Number: one or several.

Occasionally malignant change occurs.

TREATMENT

Excision, to include the epithelial wall, otherwise the cyst will probably reform.

SWEAT GLANDS

Sweat glands occur all over the body, except on the margins of the lips, the glans penis, and the inner surface of the prepuce. They are most numerous on the palms and soles. For fuller details, see Chapter 1, p. 3.

Hyperidrosis

This means an excessive production of sweat. It may be idiopathic, or symptomatic of other conditions, generalized or localized, and bilaterally or unilaterally distributed.

CAUSE

When symptomatic it may be due to hyperthyroidism, diabetes mellitus, tuberculosis, malaria, or organic disease of the central nervous system. Anxiety states and neuroses cause hyperidrosis, and many patients suffer from it in the presence of a doctor. It is also provoked by warmth, alcohol, and aspirin. In some cases, it is a familial disorder.

CLINICAL FEATURES

The patient may complain of intense discomfort from excessive sweating of the hands or feet, so much so in some cases that ability to work is impaired.

The skin of the palms and soles becomes thickened and develops a bluish-grey colour. Nail deformities may occur.

TREATMENT

Any underlying cause must be treated. When functional, the following therapies may be tried:

1. Wear only cotton socks and change daily. The same applies to underwear, when necessary.
2. Formaldehyde 2 per cent in water, dabbed on 2 or 3 times a week, not forgetting that formaldehyde is capable of ultimately causing dermatitis.
3. Hexametaphosphate 5 per cent in water, applied daily.
4. Sympathectomy is useful for severe hyperidrosis of the palms, but not for the soles.

5. X-rays should not be used.
6. For axillary hyperidrosis, the hair should be kept shaven, and anti-perspirant lotions used.

MUCOUS MEMBRANES

Introduction

This section deals only with those mucous membranes which are adjacent to the skin, namely, the oral, nasal and conjunctival, the penile, vulvar, vaginal and anal.

Although they have neither a horny layer, hairs, nor sweat glands (in the mouth especially, however, there are mucous glands), the mucous membranes are subject to a great many disorders to which the skin is also liable and in the same way; they are, for example, prone to allergic, infective and malignant processes.

In quite a number of skin conditions, mucous membrane lesions co-exist, and therefore examination of these surfaces must not be omitted. Sometimes they will enable one to confirm an otherwise difficult differential diagnosis.

It is profitable to acquire the habit of examining the mouth, in the general examination of a case, so that one does not omit the examination when a disease with which oral lesions are associated presents itself. Even when oral lesions exist, recognition is not always straightforward, for the uncornified fragile mucous membrane is soon broken and macerated by the evolution of the lesion. It is important, therefore, to look for the early unaltered lesion.

Oral examination refers to the lips, tongue, palate and gums. Lesions on these areas may be part of a disease localized to the mucous membranes; they may also exist in the presence of skin lesions; and they may be the forerunners of a skin eruption. This precept also applies to mucous membrane lesions in other sites, although in other sites lesions are, as a rule, more likely to exist without skin lesions.

The lesions associated with skin diseases are described under the named disease. They are lichen planus, syphilis, erythema multiforme and pemphigus. Other less common diseases are also associated with oral lesions.

ORAL LESIONS

The Lips

Cheilitis

This means inflammation of the lips, and is characterized by scaling, crusting, and fissuring.

CAUSE

Chemical agents. Lipsticks, toothpastes and mouthwashes.

Physical agents. Sunlight sensitivity.

Trauma. Habitual licking of the lips.

Infections. Moniliasis is the usual cause in this case.

Secondary to skin conditions, such as seborrhoeic dermatitis, or atopic dermatitis.

Drugs. Antibiotics.

TREATMENT

Removal and/or treatment of the cause. Local steroids always help except in the case of moniliasis, when nystatin cream should be applied.

Perlèche

This is an inflammatory condition at the junctures where the lips meet, and is characterized by fissuring, maceration and crusting of the area.

CAUSE

Candida albicans, streptococcal or staphylococcal infection. These may be transmitted directly, or by the communal use of cups and towels, in closed communities such as schools. Infection may also develop in people with badly-fitting dentures, which may alter the angle where the lips meet, thus creating a warm moist folded area of skin suitable for the growth of organisms.

TREATMENT

Avoid communal use of cups and towels. Faulty dentures should be corrected. Apply a steroid ointment combined with an antibiotic, or nystatin ointment when *Candida albicans* is present.

The Mouth

Aphthous Stomatitis

This is a recurrent vesicular condition of the mouth.

CAUSE

This is unknown. Some cases are associated with emotional stress, others with gastro-intestinal disturbances.

CLINICAL FEATURES

An extremely sore mouth, which induces apprehension regarding eating and drinking, because of the accentuation of the soreness.

The onset is sudden. Lesions are vesicular. They are very small, yellowish, and soon ulcerate. They are found on the side of the tongue, or its under-surface, on the inner surface of the lips and the gums. Occasionally the genitalia are affected.

COURSE

Lesions are recurrent, taking about 2 weeks to heal. The intervals between attacks vary from years to weeks.

DIAGNOSIS

Moniliasis is excluded by the absence of organisms, and the absence of foetor, fever and lymphadenopathy precludes Vincent's angina.

TREATMENT

There is nothing specific. Hot food and drink, which worsens the condition, must be avoided. Mouth-washes and the application of 1 per cent silver nitrate may help.

Leucoplakia

This condition is characterized by white patches on the lips and on any of the mucous membranes of the body. It is a potentially malignant disease.

CAUSE

This is unknown, except when syphilis is present.

Predisposing factors

Any form of chronic irritation such as smoking or jagged teeth may produce oral lesions. Blond-skinned people are more liable to suffer, whilst males far outnumber females.

CLINICAL FEATURES

At the onset there is slight irritation of the area, associated with marked sensitivity to hot and spicy foods and drinks. The lesions are flat at the onset, later becoming slightly raised. They may be very small, or the size of a sixpence, or larger. They vary in shape, being usually roughly round, but may appear as streaks, bands or nodules. They are always well defined, although the edge is irregular. The surface is flat and smooth and, as the name implies, the lesions are white. To the touch, they are somewhat rough, and may feel thickened. They are most commonly found on the dorsum and lateral sulci of the tongue, the inner surfaces of the cheeks along the interdental line, and the gums, especially at the angle of the jaw.

On the female genitalia, the clitoris, the inner surfaces of the labia and the perineum are most commonly affected; in the male, the glans penis. When lesions are present on the genitalia, itching may be very severe, although intermittent.

COURSE

When the condition affects the mouth, cessation of smoking, attention to teeth or dentures, and the avoidance of stimulating food and drink, usually result in the disappearance of the lesions. Should no improvement occur within two months, surgical treatment must be given.

Vulval lesions become malignant in 50 per cent of cases.

DIAGNOSIS

This is made by the characteristic whiteness, and the irregular and well-defined lesions. *Syphilis* can be excluded by other signs of the disease, and *lichen planus* by lesions characteristic of that disease found elsewhere in the body.

TREATMENT

Cauterization or excision of oral lesions is best, whilst vulvectomy is essential for vulval lesions.

NAILS

The anatomy and pathology of the nails, and other aspects, are considered in Chapter 1.

Nail affections may be primary, or secondary to other conditions, both dermatological and non-dermatological.

PRIMARY CONDITIONS

1. Ringworm

and other fungal infections of the nails are dealt with in Chapter 13.

2. Paronychia

This is an acute or subacute inflammation of the peri-ungual tissues of one or more nails (Fig. 53).



FIG. 53. Paronychia showing bolstering of the nail-folds, and deformity of the nails (Institute of Dermatology, University of London).

CAUSE

Usually *Candida albicans*, occasionally streptococci or staphylococci.

Sex

Women more often affected than men.

Occupation

Bar-work, laundry-work, kitchen-work, manicuring; for maceration of the skin due to excessive immersion in liquids facilitates the entry of organisms under the nail-fold.

CLINICAL FEATURES

Pain localized to the peri-ungual tissues. The peri-ungual tissues of one or more digits is swollen, red, macerated, and painful to the touch. The middle finger is most commonly affected first. Gentle pressure on the bolstered nail-fold may result in the expression of a bead of pus.

TREATMENT

The most important factor is to keep the finger dry. If no organisms can be cultured, nystatin ointment should first be used. It is gently inserted on a finely-pared, square-ended orange stick, under the nail-fold, twice a day. Should no improvement occur after 2 to 3 weeks, eusol lotion, or Penotrane jelly, may be applied in the same way.

In the most obstinate cases, X-ray treatment may be required. Avulsion of the nail is not recommended.

PROGNOSIS

Most cases clear up in 4-8 weeks, but recurrences are not uncommon. These can be minimized by the avoidance of excessive washing, and protecting the finger with a rubber stall whilst following an occupation demanding immersion in liquids.

3. Ingrowing Nail

In this condition the lateral border of the toenail grows into the surrounding soft tissues.

CAUSE

Tight shoes, usually.

TREATMENT

Spacious shoes. Also cut a V into the middle of the free edge of the nail. Relief of pain may be obtained by the daily application of cold Kaolin cream on lint.

4. Hypertrophy of the Nail (Onychogryphosis)

The toe-nails become enormous, deformed and filthy, the condition being commonly due to neglect and badly-fitting shoes. It is common in vagrants.

SECONDARY CONDITIONS

1. Clubbing of the Fingers

Known also as Hippocratic fingers, this results in enlargement and curving in both directions of the finger, and/or toe-nails.

They are due to chronic lung diseases, such as bronchiectasis, chronic heart disease, such as congenital heart disease, or sub-acute bacterial endocarditis, and occasionally chronic gastro-intestinal disease. In about 10 per cent of cases the cause is familial.

2. Spoon-shaped Nails, or Koilonychia

May be congenital in origin, or due to the Plummer-Vinson syndrome. Occasionally they have been noted in coronary artery disease, syphilis, and in users of strong alkalis. The nails are so shaped that if a drop of water is placed on them it will not roll off.

3. Leuconychia

These are white spots or streaks on the nails, and are usually congenital or due to trauma. They may also appear spontaneously, as a result of air in the nail.

4. Brittleness of the Nails

May be congenital or acquired. When acquired, it is due to detergents, nail polish or polish removers, myxoedema, or old age.

5. Shedding of Nails

This sometimes accompanies alopecia areata, and is a minor complication of infectious fevers, such as typhoid and meningitis. The nails ultimately grow again, except in severe peripheral vascular disease.

6. Pigmentation of the Nails

May be due to the use of potassium permanganate as an antiseptic finger bath, or the taking of phenolphthalein, a constituent of some laxatives, or mepacrine which is used in cases of chronic lupus erythematosus and malaria.

7. Nail Changes in Skin Diseases

are dealt with elsewhere in the description of the disease but a summary is given in Table 3.

TABLE 3

Nail changes which may occur, singly or severally in some skin diseases

PSORIASIS	'Thimble pitting', yellowish discoloration, subungual accumulation of horny material
ECZEMA	Irregularity, ridging
CHRONIC PARONYCHIA	Distortion and ridging
ALOPECIA AREATA	Temporary arrest of growth, or fall
ICHTHYOSIS	Discoloration, subungual thickening
LICHEN PLANUS	Discoloration, ridging, and shedding

HAIR

The anatomy, function and other aspects of the hair, are dealt with in Chapter 1.

ALOPECIA

Alopecia, or baldness, may be congenital or acquired, congenital baldness being very uncommon. Consideration of any form of

alopecia must embrace all the hairy areas of the body. The scalp is most commonly affected, but involvement of the groins, beard, axillae, and pubes must be borne in mind.

Congenital Alopecia

This may be complete, permanent, and generalized, or variations of these states may occur. For example, there may be complete or partial alopecia followed later by the development of normal growth.

Congenital alopecia is frequently associated with abnormal growth, or absence of teeth or nails, and skin irregularities. It is worth remembering that where one congenital anomaly occurs, others frequently exist in other organs and tissues.

CAUSE

Unknown.

Sex

The condition is more common in men.

Heredity

More often than not, no relevant history can be elicited.

Treatment

None effective.

Prognosis

This is impossible to forecast, and as a rule no growth should be expected.

Acquired Alopecia

Common Baldness

This is the commonest form of baldness, and accounts for at least 95 per cent of all cases.

CAUSE

Unknown.

Theories

The most popular current theories vacillate between genetic, hormonal, or ageing factors as being responsible, severally or singly.

Sex

Men far outnumber women, as is obvious from daily observation.

CLINICAL FEATURES

The onset is gradual. The baldness is never scattered, and pursues a well-recognized course, producing one of the common types of male baldness. Recession may occur in the fronto-parietal areas, or on the crown of the head, or on both regions simultaneously. The rate of hair-fall varies in each person, and cannot be anticipated.

DIAGNOSIS

This is made by the pattern of recession, and the absence of broken hairs (see alopecia areata).

TREATMENT

There is no effective prophylactic or curative treatment known. Promises made by quack hair-restorers are baseless.

Alopecia Areata

This form of alopecia is characterized by a loss of hair in round or oval, well-defined patches, without inflammation.

CAUSE

Unknown.

Sex

Both sexes are equally affected.

Age

Any age may be affected, but the commonest is childhood. At least 20 per cent occur before the age of ten.

Heredity

It has often occurred in several members of a family. There are also reports of identical cases in twins. An hereditary history in at least 20 per cent of a series of cases is not unusual.

Psychological Factors

Much has been written in favour of and against the involvement of such factors in alopecia areata. There are many cases of the condition being coincidental, for example, with bereavements of a close relation, the ordeals of domestic strife, or the unfulfilment of ambition; there are also many cases with no apparent emotional disturbance whatsoever, so that it is difficult to be dogmatic. Each case must be judged on its merits.

Other theories

Virus infections and hormonal disturbances have been blamed, but such assumptions have yet to be proven.

CLINICAL FEATURES

There are no symptoms.

The onset is nearly always sudden. In some cases, an area may become completely bald overnight, the patient finding a cluster of hairs on the pillow. In other cases, baldness develops over the course of a few days. The size of the area varies from that of a sixpence to the palm of the hand, or larger (Fig. 54). Their shape is generally round, but may be oval, or very irregular due to coalescence of patches. The surface of the scalp is white, soft, and smooth; and close examination will reveal very short broken stumps of hair, which are clearly thicker at the top than at the skin surface, illustrating a characteristic feature of the disease, namely exclamation-mark hair, thus, !. When they disappear, arrest of the balding process has begun. Any site on the scalp may be involved. Any hairs remaining may be easily removed, and whilst the patch is active, long hairs at the edge are easily pulled out with their shiny root-sheaths. Once activity ceases, the hairs are firm. The eyebrows, beard, moustache and eyelashes are other areas which may be involved. The number of patches ranges from one to many.

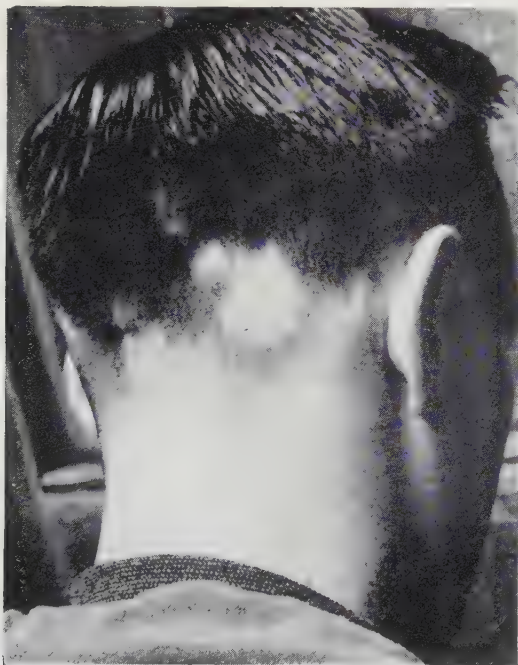


FIG. 54. Alopecia areata. Partial scalp involvement.

COURSE

Regrowth is not a certainty, but usual. As regrowth occurs, the hairs appear as soft down, and are soon replaced by thicker white hairs, which are later replaced again by pigmented normal hairs. During these phases, a piebald appearance may be expected.

Variations of Alopecia

- (i) *Alopecia totalis*: signifies loss of hair over the entire scalp (Fig. 55).
- (ii) *Alopecia universalis*: loss of hair over the entire body.

(iii) *Traction alopecia*: is found on the frontal and occipital areas, as a result of the hair being pulled when the 'pony tail' style is worn, or when pulled by the use of tight curlers.



FIG. 55. Alopecia areata. Total scalp involvement.

TREATMENT

Although there is nothing specific some mild counter-irritant should be given, and attention paid to the general health of the patient.

External

Painting the area with 1 or 2 per cent phenol by means of a used toothbrush, every 2 days, or Thorium X varnish every 2 weeks is

sometimes useful. One injection of a steroid preparation into small patches is often enough to initiate regrowth, although more than one injection may be necessary. (Steroids are useless for male pattern baldness.)

Internal

Tonics and iron when necessary may be helpful.

DIAGNOSIS

This is made by the history of sudden hair fall in patches, the scalp being normal except for exclamation-mark and loose hairs.

PROGNOSIS

The younger the patient the better the outlook. The older patient is always very anxious concerning regrowth and the possibility of complete baldness, and therefore the prognosis often depends to a large extent on the enthusiasm and confidence of the doctor. Intralesional steroids have greatly improved the prognosis, although they are hardly justified in children.

HIRSUTIES

Hirsuties is also known by the term hypertrichosis, both words signifying superfluous hair. This is found on areas which naturally have downy hair. It may be congenital or acquired.

Sex

It is found equally in both sexes, but it is women who seek medical advice because of its embarrassment to them.

Race

There is a tendency for the condition to be more common amongst Jews, negroes and Spaniards.

Hormonal disturbances

Pregnancy is sometimes accompanied by sudden hirsuties, which is usually transitory. The menopause is also sometimes associated with the gradual appearance of superfluous hair, which is, however, permanent.

Other conditions, such as Cushing's syndrome, the adreno-genital syndrome, acromegaly, tumours of the pineal gland, and hyperthyroidism must also be considered as causes of hirsuties.

Psychological factors

Anorexia nervosa is always accompanied by hirsuties of the downy type, although some coarse hairs may be seen.

Drugs

Steroids may produce hirsuties.

CLINICAL FEATURES

In women, mental distress is the predominant feature.

(a) *Congenital type*. In severe cases, the entire body and face may be covered with hair. Persons so afflicted may earn a living working in travelling circuses. A lock of hair confined to the lumbo-sacral area may be associated with spina bifida. Other anomalies distributed on small areas of the body may be found. Dental defects may be found with the localized variety of hirsuties.

(b) *Acquired type*. Where there is no obvious cause, in the case of a healthy woman, the superfluous hair is always localized, and found on the beard area. Psychoses may develop.

When hirsuties is due to a primary condition, for example, Cushing's syndrome, that condition will clearly declare itself as the cause.

In children, transient hirsuties may be seen, varying in magnitude and distribution, in such conditions as tuberculosis, coeliac disease, malnutrition or pink disease.

TREATMENT

Temporary removal can be obtained by the judicious use of a pumice stone, by shaving, or better still, by waxes. The wax is applied soft and warm, and when cool is rapidly removed pulling with it attached hairs. This operation is repeated every 3 or 4 weeks. None of these methods increase hair growth. For less exuberant hirsuties, bleaching by means of hydrogen peroxide with cotton wool once or twice a week disguises the condition.

Permanent removal is obtained by using a diathermy needle, which is inserted into the hair follicle.

ANOMALIES OF HAIR-GROWTH

Some of the many anomalies are worth mentioning.

1. Greying of the hair

This is very common, and the cause is unknown. A familial tendency is probably the commonest factor.

2. Ingrowing hairs, or pili incarnati

These hairs turn round and re-enter the skin, after surfacing. The beard area is commonly affected; when the eyelashes are concerned, conjunctivitis may occur as the hair is in the process of turning on itself. Growing a beard is sometimes effective in training the hair to grow outwards. For eyelashes, epilation should be used.

3. Monilethrix

A congenital and hereditary condition, characterized by dull, thin, brittle hair on the scalp, with beaded swellings of the hair shaft. Both sexes are affected. Normal growth sometimes occurs spontaneously at puberty.

CHAPTER 19

Tumours

Introduction

A tumour is a mass, and in the lay mind is *ipso facto* considered to be malignant.

Tumours of the skin, like tumours of any other tissue, can, of course, be benign or malignant.

By definition, their characteristics in the skin vary enormously, being soft or hard, freely mobile or fixed to the surrounding tissue, being raised above the skin surface or situated deep in it, whilst their shape and size know no uniformity.

The commonest malignant conditions of the skin are the basal cell and the squamous cell carcinoma, and the former is more prevalent than the latter. But by and large, masses in or on the skin, in this country, are more often benign than malignant.

The most heartening feature of the basal cell carcinoma is that it very, very rarely spreads systematically through the body, although the squamous cell carcinoma will do so if not dealt with early enough.

Some benign tumours, such as haemangiomata or birthmarks, and cellular naevi or moles, are dealt with in Chapter 20.

BENIGN TUMOURS

Seborrhoeic Warts (Senile Warts)

The title is misleading, for these lesions are not warts in the sense that they are caused by a virus, nor are they, therefore, infectious (the cause is unknown), but in appearance they are what one recognizes as warty. Nor are they associated with seborrhoea. And again,

senility is not an essential condition for their existence. Such is the confusion of nomenclature. Although they are commonly found in old people, they may develop at the age of forty. Nevertheless, seborrhoeic is a better word to use than senile in the patient's hearing.

PATHOLOGY

Hyperkeratosis, acanthosis and papillomatosis are apparent, and the basal layer is notably hyperpigmented. The pigmentation varies in density, which accounts for the different shades of brown of the clinical lesion.

CLINICAL FEATURES

The lesions are papular, the size varying from 1–2 cms. in diameter. They are round or oval, the surface is rough and flat, and their colour varies from light to dark brown (Plate 27). The commonest site is the trunk, and they also occur on the temples and elsewhere. Sometimes a few exist, at other times they are scattered haphazardly over the trunk.

DIAGNOSIS

They must be distinguished from pigmented moles which are very smooth, well defined, and appear earlier in life.

TREATMENT

Curetting followed by cauterization, or cauterization alone.

Keloid

This is a firm, irregularly-shaped tumour, which may be smooth or ridged, occurring generally on the area of a scar or a previous injury.

CAUSE

- (a) An individual predisposition.
- (b) A racial predisposition, especially in negroes.
- (c) A regional susceptibility, in that keloids commonly occur in areas where an injury crosses normal flexion creases.
- (d) Trauma, which, however slight, may initiate the formation of a keloid in susceptible people.

PATHOLOGY

The collagenous tissue of the dermis is enormously increased and obliterates the elastic tissue and, partially, the blood vessels in the area. This increase of collagen accounts for the tumour formation.

CLINICAL FEATURES

The onset is gradual. The lesions are smooth or ridged swellings. Their size varies from a pip to a plate (Fig. 56), and any kind of



FIG. 56. Keloid, which followed a burn from boiling water.

shape may develop, such as a cord, lozenge or spider. The surface is glossy and smooth, and the colour which is at first red, becomes pink, and finally white with age. Any site may be affected.

TREATMENT

Fresh lesions are best treated with X-rays followed by excision. Injections of hydrocortisone are sometimes useful.

Old lesions may be treated by these methods too, but the results are poor.

Fibroma

This is a hard, painless, well-defined nodule, usually found on the extremities, in adults. In some cases, multiple lesions exist. The cause is not known.

PATHOLOGY

Two types exist; in one, the chief cells are histiocytes, in the other fibroblasts. The lesion is well defined but does not have a capsule. The clear definition is due to a band of normal collagen. The lesion contains innumerable histiocytes, which contain lipoids, and giant cells are also present.

CLINICAL FEATURES

The onset is quite rapid. The lesion becomes akin to an acorn's nut, shiny and firm, with epidermis very adherent to it.

TREATMENT

Excision, if required.

Neurofibroma

This condition is characterized by numerous, flabby, flesh-coloured or brownish pedunculated tumours, and brown macules (Fig. 57).

There are 3 types:

(i) Superficial, which is the type most commonly seen in skin clinics.

(ii) Deep, where the lesions are attached to the peripheral parts of nerve trunks.

(iii) Multiple neurofibromatosis, or von Recklinghausen's disease.



FIG. 57. Neurofibromatosis.

PATHOLOGY

The tumour is comparatively acellular, and is composed of wavy bundles of fibroblastic cells, with long thin nuclei separated by loose connective tissue, in which groups of nerve fibres are easily visible.

CLINICAL FEATURES

(1) Superficial type: these are pin-head in size, occasionally becoming as big as a nut, and commonly occur during pregnancy.

(2) Deep type: these can be felt along the main trunks of nerves, for example, the brachial.

(3) Multiple: brown macules, also called *café-au-lait* spots, may occur without any tumours. But they may also occur in association with (a) tumours in the skin, or (b) enormous, pendulous, pigmented tumours, or very widespread pigmented lesions covering the bathing-suit area.

Central nervous system abnormalities and bony changes also occur.

DIAGNOSIS

This is made by the *café-au-lait* spots and the pedunculation of the lesions.

TREATMENT

Excision when required.

Lipoma

These are soft rounded swellings occurring in the dermis and/or subcutaneous tissue, singly or severally, and as their name suggests, they are composed of fat cells.

They are usually found on the shoulders and back, and sometimes cause discomfort to their owner when supine.

TREATMENT

is by excision.

Senile Keratosis

This condition occurs as a horny lesion, on areas exposed to sunlight, in elderly people. Malignant change occurs in about 20 per cent of cases.

PATHOLOGY

Hyperkeratosis, acanthosis, papillomatosis and a chronic inflammatory infiltrate in the dermis can be seen.

Evidence of malignant change must be excluded.



FIG. 58. Senile keratoses (Dr E. Waddington).

CLINICAL FEATURES

The lesions are papular, and vary in size from a pin-head to a coin (Fig. 58). The shape is variable, and the outline well defined. The surface is rough and horny, and when removed bleeding occurs. Sometimes the scale is heaped up in a lump, at other times it is thin and wafer-like; in both instances it is quite adherent. The colour of the lesion is brownish. When there is inflammation around the edge, malignant change must be considered.

DIAGNOSIS

The condition must be distinguished from senile warts, which are soft and not covered with a scale.

TREATMENT

Cauterization, superficially applied, is usually quite sufficient for a good result.

Granuloma Pyogenicum (Granuloma telangiectaticum)

This occurs as a single, soft, raspberry-like tumour, at any age, in either sex. The cause is unknown, but it is often preceded by minor trauma, such as a penetration of the skin by a thorn.

PATHOLOGY

The epidermis is very thin, because of the great number of newly-formed and dilated capillaries which press up against the epidermis, flattening it.

CLINICAL FEATURES

The onset is sudden. The lesion is nodular, and pea to cherry size (Fig. 59). The surface is smooth and glistening, although some areas may be slightly ulcerated. It looks like an over-ripe fruit. The colour is bright red (Plate 28), due to the excess of capillaries within. Sites commonly affected are the finger, hand, leg, or back. The lesion bleeds easily, and may alarm the patient.

DIAGNOSIS

This is made by the rapid onset, and the tendency to bleeding.



FIG. 59. Granuloma pyogenicum (Institute of Dermatology, University of London).

TREATMENT

Excision followed by cauterization of bleeding surfaces.

PROGNOSIS

The tumour may recur, however skilful the operation.

Kerato-Acanthoma

Molluscum Sebaceum

This term describes a cherry-sized, shell-like tumour, which usually occurs in adults, and in males more often than females. It undergoes spontaneous involution within several months, leaving a depressed and atrophic scar. The cause is unknown.

PATHOLOGY

The microscopical appearance is so like that of a low-grade squamous-cell carcinoma, that differentiation is often impossible.

In this condition, however, the cell changes of carcinoma are absent, and there is a very limited degree of invasiveness.

CLINICAL FEATURES

The onset is quite rapid, and full size is attained in a few weeks. The lesion is round and firm, with a central crater containing horny material (Plate 29). The lesion may grow to a size of 2 or 3 inches, remains well demarcated, and seems to be stuck on the skin. It is flesh-coloured. Commonest sites are the face, backs of the hands and forearms.

TREATMENT

Curettage followed by cauterization is best.

Leucoplakia

This is simply a hyperkeratosis occurring on a mucous membrane (Fig. 60) and is discussed on page 196.

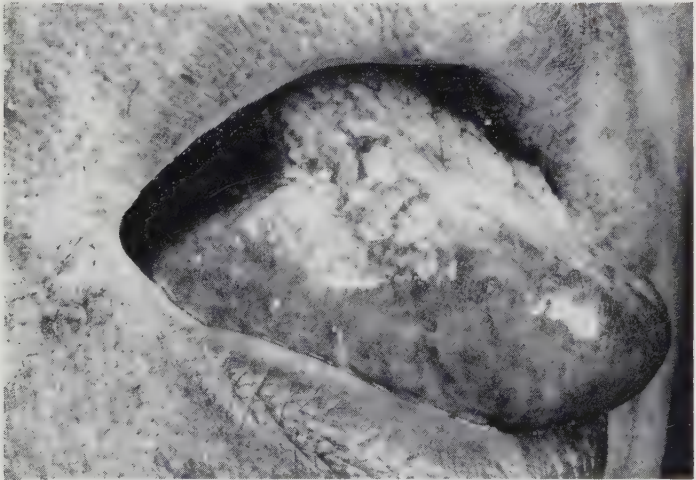


FIG. 60. Leucoplakia. Sharply defined irregular white patches (Institute of Dermatology, University of London).

P*

MALIGNANT TUMOURS**Basal Cell Carcinoma***Rodent ulcer, or basal cell epithelioma*

The condition to be described is best known by the three names above. None of them is absolutely correct. Although most of these tumours arise from basal cells, some few do not. Rodent ulcer is the oldest of the synonyms, but many of these tumours do not ulcerate. An epithelioma means any tumour derived from the epithelium.

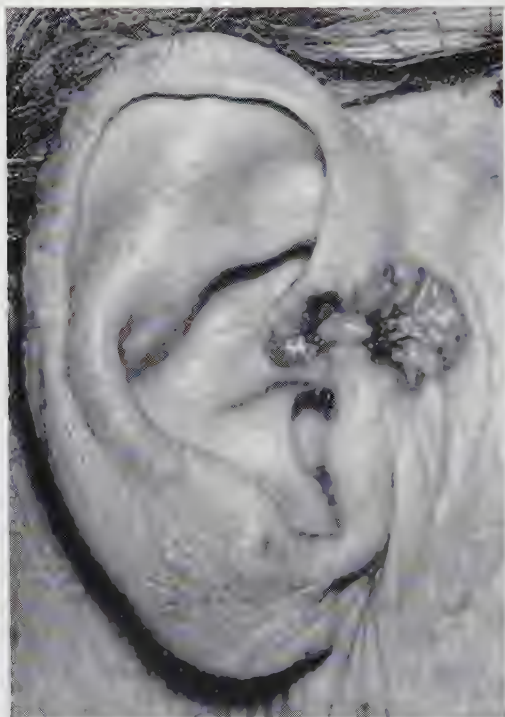


FIG. 61. Basal cell carcinoma (Department of Dermatology, Addenbrooke's Hospital).

Whichever name is preferred, the lesions are recognized as superficial or deep nodules or indurated ulcers, usually on the face, but sometimes elsewhere. They rarely metastasize, but ulcerate the surrounding tissue in which they lie.

They are uncommon before the age of 40, and males are much more prone than females.

PATHOLOGY

The tumour cells are very characteristic. They are large, oval, and stain deep blue-black with haematoxylin. Although they may appear

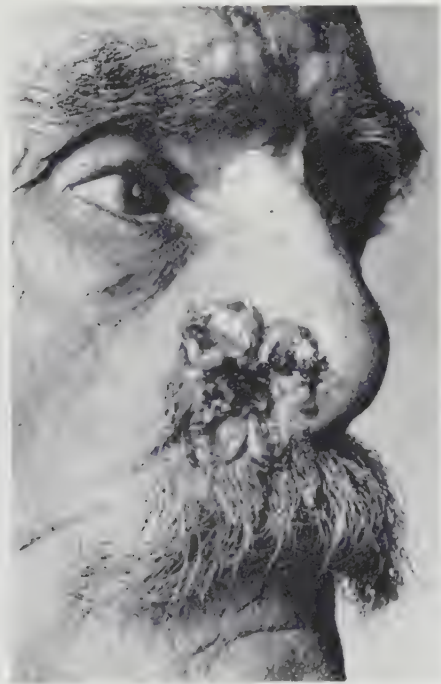


FIG. 62. Basal cell carcinoma (Dr E. Waddington).

in the epidermis, they commonly lie in various groups in the dermis, the cells inside the group being haphazardly arranged, whilst those all around the edge of the group are arranged as a palisade.

An inflammatory reaction in the dermis varies according to the rate of growth of the tumour, being more pronounced in rapidly growing lesions.

CLINICAL SIGNS

The onset is insidious. The lesion is nodular, growing from pin-head to pea-size, or somewhat larger. The edge is pearly, shiny and raised, and this is a notable characteristic. The surface of the lesion may be unbroken, or ulcerated. When the crust is removed bleeding easily occurs. Commonest sites are the face (Figs. 61, 62) and forehead (Fig. 63), but the scalp (Fig. 64), forearms, or trunk (Fig. 65) may become involved.

There are three common clinical types of lesion. 1. Button-like, or nodulo-ulcerative. 2. Pigmented nodulo-ulcerative. 3. Fibrotic

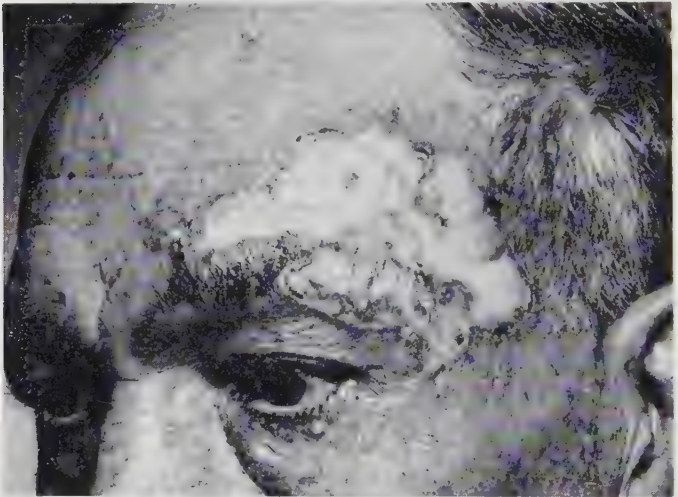


FIG. 63. Basal cell carcinoma, showing a marked rolled edge (Dr E. Waddington).

type, appearing as a slightly-raised, firm, yellowish plaque, which usually ulcerates.



FIG. 64. Basal cell carcinoma of the scalp.

DIAGNOSIS

This is made by the long history of slow growth, and the appearance of the pearly-edged nodule. A biopsy will, of course, clinch the diagnosis.

A rapid method of confirming a clinical diagnosis is by cyto-diagnosis, a manoeuvre in which one scrapes the ulcerated area,

applies the tissue to a slide, and stains with haematoxylin and eosin. A positive result will show numbers of the cells peculiar to this condition.

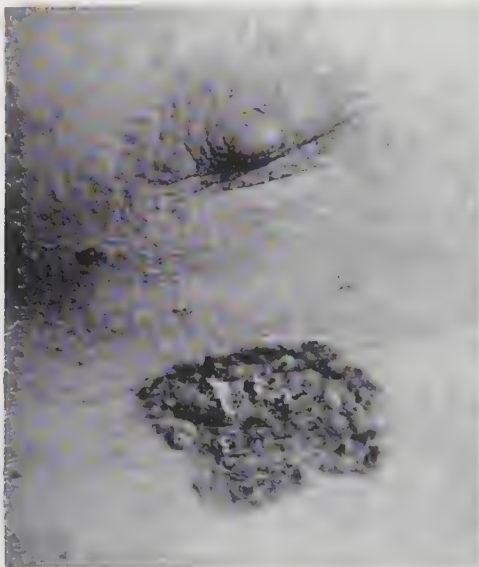


FIG. 65. Basal cell carcinoma (Dr E. Waddington).

DIAGNOSIS

This is made by the long history, slow growth, pearly and waxy edge, and biopsy. Squamous cell carcinoma grows faster, sometimes half an inch in 3 months, and is white and opaque. Kerato-acanthoma grows rapidly, stops growing within 4-8 weeks, and does not ulcerate.

TREATMENT

Excision, X-rays, cautery, or diathermy.

PROGNOSIS

A small percentage recur, however effective the treatment seems to have been.



PLATE 28. Granuloma pyogenicum (Dr Martin Beare).

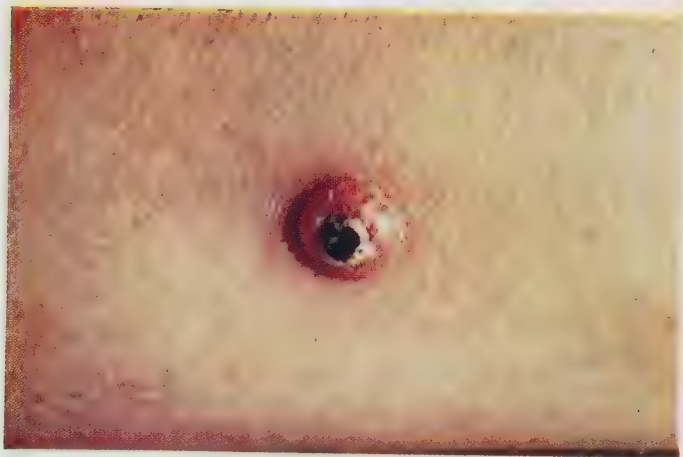


PLATE 29. Kerato-acanthoma; fully-developed lesion with central crater.

PLATE 30. Paget's disease
of the nipple, indurated
and infiltrated with
destruction of the nipple.



PLATE 31. Haemangioma
(Dr G. A. Hodgson).



Squamous Cell Carcinoma

This differs greatly from the basal cell carcinoma, being a true invasive tumour, which develops in normal tissue, or in a pre-existing lesion, such as leucoplakia, or senile keratosis. It commonly occurs in elderly people.

PATHOLOGY

Masses of irregularly grouped epithelial cells teem downwards to invade the dermis. These masses contain flat prickly cells very distorted in size and shape, intercellular fibrils, and, dotted about, shiny horny pearls. In reality, they are composed of concentrically arranged layers of horn cells enclosing a central area of keratinization, or horn.

In rapidly growing lesions there are many atypical cells, as well as mitoses. The degree of malignancy can be estimated by consideration of the ratio of the differentiated to undifferentiated cells.

CLINICAL FEATURES

The onset is gradual. The lesions may begin as hard nodules, sometimes with a warty surface; or as flat scaly indurated lesions. When the growth is 1 or 2 cm in diameter, ulceration occurs, and the edge of the ulcer becomes thick and everted. The surface may be papillomatous, or like a cauliflower, and have a very smelly exudate.

Occasionally, the lesions do not ulcerate. Localized lymphadenitis may be present, and metastases should be sought.

Ulcerative lesions fluoresce orange under Woods light (see p. 121).

DIAGNOSIS

This is made by the comparatively rapid growth, the marked induration, and biopsy, or cytodiagnosis.

TREATMENT

Excision is the best method for most lesions. X-rays give good results for lesions under an inch in diameter. Lymph nodes must be treated when necessary. The patient must be regularly examined for 5 years, as if a relapse occurs it is normal between 2 and 3 years.

Paget's Disease of the Nipple

Carcinoma of the Nipple

This is a rare tumour, which involves the female nipple or areola, and is nearly always unilateral. It normally occurs between the ages of 40 and 60. It is sometimes confused with eczema.

PATHOLOGY

Paget cells are large, and easily visible in the epidermis. Around them is a clear space. Their nuclei are large, round and pale. Early lesions often fail to show Paget cells. There is also a marked inflammatory infiltrate.

CLINICAL FEATURES

The onset is insidious. The early lesion is papular, and round and well defined. At first small, it enlarges up to palm size. It is red and the surface is scaly, which when removed reveals red, eroded or oozing surfaces. In this state it is very like eczema.

The lesion gradually becomes indurated, infiltrated and sometimes ulcerated. The nipple may become retracted or destroyed (Plate 30).

Untreated, the lesion is relentlessly progressive, and lymph node enlargement and metastases are common in advanced cases.

DIAGNOSIS

This is made by its unilateral distribution, its induration and chronicity, and failure to respond to simple remedies.

It must be distinguished from *eczema* of the nipple, which is normally bilateral and in which the lesion is soft and responds to simple measures.

TREATMENT

Radical mastectomy, and removal of axillary lymph nodes, followed by X-rays, is the method of choice.

EXTRA-MAMMARY PAGET'S DISEASE

is found on the vulva and peri-anal areas. The mammary gland is a modified sweat gland, and extra-mammary lesions are considered to be intra-epidermal metastases from an apocrine gland carcinoma. Systemic metastases are rare.

Mycosis Fungoides

This is an uncommon, chronic and invariably fatal disease, which is accompanied and preceded by severe itching. It only affects adults, and rarely females.

The name is misleading as it has nothing to do with fungus infection, but is a form of reticulosis.

CLINICAL FEATURES

There are 3 clear-cut stages:

1. Eczematous, which lasts a few months or years.
2. Infiltrative plaque, often accompanied by exfoliative dermatitis. In this stage death may occur, or the disease may continue into the third stage.
3. Tumour; these infiltrated thick lesions break down to form deep sloughing ulcers. Death follows from exhaustion within a few months or years.

Duration

Death occurs within 5 years of the onset, unless treatment is given.

TREATMENT

X-rays are the most useful form of treatment, and at best only produce temporary remission.

Malignant Melanoma

This rare tumour arises from a pigmented naevus (p. 238), or apparently normal skin, and is characterized by a blue-black nodule which gradually increases in size.

It occurs at any age after puberty, and the sexes are equally affected.

PATHOLOGY

The malignant changes almost always occur at the epidermo-dermal junction. The naevus cells already there, as part of a pigmented naevus, suddenly become bloated, and take in more melanin, so that they stain very darkly. The cell nuclei also enlarge, and mitosis is seen. Soon the cells proliferate and flood into the dermis and epidermis, and evidence of a breakthrough into the lymphatics and blood vessels may be noted.

CLINICAL FEATURES

The onset is insidious, or quite rapid. The lesion becomes nodular, as it grows up to the size of 2 cm on a pigmented naevus. The colour is most striking being blue-black, or jet black, which the patient will comment on, and at the edge of the lesion a red halo may be seen. The sites commonly affected are the head and neck (Fig. 66), and then the lower extremities, but no area is exempt, a melanotic whitlow being one of the more unusual sites. Lymphadenopathy occurs early, and melanuria, melanaemia, and wasting soon follow.

The growth of a malignant melanoma is increased by pregnancy.



FIG. 66. Melanoma (Institute of Dermatology, University of London).

DIAGNOSIS

This is made by the sudden increase in size and depth of colour of a pigmented naevus, and by bleeding and ulceration of the lesion.

When the tumour arises from normal skin, a small black or brown spot appears followed by rapid growth.

The diagnosis must be made from dark haemangiomas, traumatic blood blisters and pigmented basal-cell carcinomas.

TREATMENT

These lesions must be treated as emergencies, and any suspected lesion should be widely and deeply excised and subjected to biopsy.

PROGNOSIS

This depends on radical surgery at the earliest possible moment.

Hodgkin's Disease

This is a systemic disease of the lymphoid-reticular system affecting primarily and predominantly lymph nodes. In a few cases, the skin may be first affected.

CLINICAL FEATURES

In most cases there is generalized itching. Urticarial, bullous, vesicular or papular eruptions occur. Alopecia and purpuric lesions may also appear, and herpes zoster as a complication should not be forgotten. Splenomegaly and pyrexia are also present.

TREATMENT

X-rays cause disappearance of the lesions which tend to recur. Nitrogen mustard, intravenously, produces temporary involution. Prednisolone or ACTH may help. Wide excision of glands is carried out in some cases.

Leukaemia

Involvement of the skin in the lymphatic, myelogenous, and monocytic forms is very similar, but is most common in the lymphatic form.

In the *chronic* leukaemias, nodules and tumours are common. In the *acute* leukaemias, purpuric, haemorrhagic bullous and ulcerative lesions are more usual.

Herpes zoster and exfoliative dermatitis (as in Hodgkin's disease) should not be forgotten as possible complications of the disease.

TREATMENT

Steroids or nitrogen mustard are used.

Metastatic Carcinoma

Cancer of the skin, secondary to a primary source in the body, is very rare. Cancer of the breast is the commonest form to metastasize in this way. Skin metastases have been seen with hypernephromas, gastric, lung and uterine cancers.

Skin Changes related to Internal Cancer

Itching may be generalized in Hodgkin's disease, lymphatic leukaemia, cancer of the breast, stomach, pancreas, lungs or prostate.

Urticaria may also occur with the above-mentioned conditions.

Flushing of the face, and upper part of the body, may occur with carcinoid tumours.

CHAPTER 20

Congenital and Hereditary Diseases

Introduction

Congenital diseases can exist at, before, or appear after birth. They may or may not be inherited; and not all inherited disorders are congenital. Fortunately, the great majority of skin diseases are rare, and many of them are collector's pieces; the minority are more common, and some of them will be described here.

For a description of the factors responsible for the appearance of congenital affections, specialized text-books should be consulted. Unlike the majority of disorders with which the dermatologist has to deal, the question of treatment of congenital skin diseases in the case of most disorders is overshadowed by the knowledge that much improvement is difficult to obtain, and in some of the more serious and rare conditions, life can only be precariously enjoyed.

The problem of classification is difficult because the clinician prefers the use of terms which describe the tissue affected by the disease, whilst the geneticist requires his own special terminology, dependent upon the transmitting factors of the disease.

Ichthyosis

This common condition is characterized by a dry, rough, and scaly skin (Fig. 67), sometimes resembling fish-scales (*ichthus*—a fish), which most severely affects the extensor surfaces, and usually spares the flexor ones.

All degrees of the condition may be seen, from an infant encased in a bone-dry suit of armour of scales, to a skin whose perfection is marred by slight scaling. This latter form is extremely common.

<i>Ectodermal Disorders</i>	<i>Mesodermal Disorders</i>	<i>Ecto- and Mesodermal Disorders</i>
*Ichthyosis	*Epidermolysis bullosa (simple and dystrophic types)	*Neurofibromatosis
Congenital ichthyosiform erythroderma		
*Keratosis of palms and soles		Acanthosis nigricans (juvenile type)
*Congenital alopecia		
*Albinism	Congenital lymphoedema	
White forelock		
Pachonychia congenita	Urticaria pigmentosa	
	*Naevus flammeus	
Congenital absence of nails	*Simple haemangioma	
Congenital antidrotic ectodermal defect	*Cavernous haemangioma	
Porphyria	Blue naevus	

N.B. Only those marked with an asterisk are described in this book.

CAUSE

Inheritance

A relevant history can nearly always be obtained.

Age

It is noticeable early in infancy, or at any time up to the age of two years.

Sex

Males are slightly more affected than females.

PATHOLOGY

Hyperkeratosis is present, which accounts for the thickening of the skin. The sweat glands are usually normal, but the sebaceous glands may be absent, or atrophic.

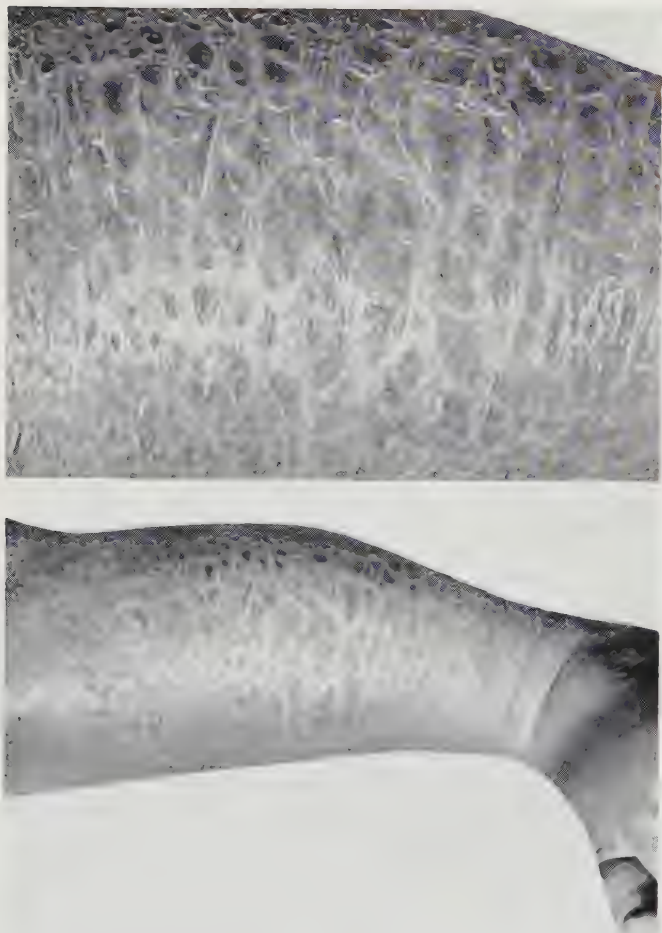


FIG 67. Ichthyosis. Rough dry scaly skin resembling fish scales.

CLINICAL FEATURES

There are no lesions, as such. Dry scaling, and thickening of the skin on the extensor surfaces predominates. The scales are centrally fixed to the skin, and are loose and slightly turned up at the edges. The tops of hair-follicles may be capped by hard keratotic papules.

The hair is thin and dry, and the nails are brittle and dry. The amount of scaling and hair and nail involvement depends on the severity of the affection.

TREATMENT

Irritants, such as detergents and soaps, must be avoided. Greasy applications such as eucerin cream, or a mixture of 3 parts olive oil to 1 part of glycerine are helpful. Bran or oatmeal baths are sometimes soothing.

A warm sunny climate is better than a cold one.

Keratosis of Palms and Soles*Tylosis*

This condition is characterized by symmetrical thickening of the skin of the palms and soles, of varying degrees. Apart from being congenital, it is also often hereditary. In some ways it appears to be related to ichthyosis.

In general, treatment should be similar to that employed for ichthyosis.

Haemangiomas

These lesions are composed of newly-formed blood vessels, and are present at birth, or appear within the first few months of life. They are very common, and there are three main types:

1. Simple haemangioma (strawberry mark).
2. Cavernous haemangioma.
3. Naevus flammeus (flat haemangioma, or port-wine stain).

Simple Haemangioma

This is a bright red raised soft tumour, often appearing like a strawberry (Fig. 68).

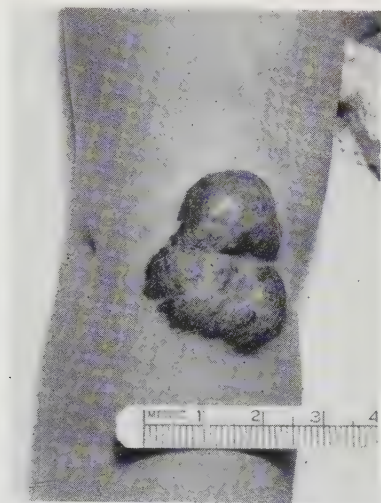


FIG. 68. Haemangioma (Dr G. A. Hodgson).

PATHOLOGY

This shows an increase and dilatation of capillaries.

CLINICAL FEATURES

The lesions vary in size, up to a medium-sized strawberry. They occur anywhere, and are generally single.

TREATMENT

None is usually required, as they involute spontaneously by the age of five years, leaving a better end result than operative measures can accomplish. Treatment is only indicated where the lesion interferes with sight or sucking.

Cavernous Haemangioma

These lesions are composed of fully mature blood vessels, and are far more solid to palpation than the simple variety. They scarcely

fade on pressure. They usually fade spontaneously by the age of 5 years.

Naevus Flammeus

This is characterized by one or several dark-red or pink patches, irregular in outline (Plate 31), and not raised above the skin.

It is formed by a diffuse telangiectasia of mature blood vessels in the dermis.

CLINICAL FEATURES

The lesions are usually unilateral on the face or neck (Fig. 69), but other areas may be affected. Lesions may be of any size. The mucous membranes may also be involved. Crying, coughing, or exposure to cold alters the lesions' colour.

TREATMENT

None is really satisfactory, but cosmetic creams can be used to hide the blemish, which is usually a great psychological handicap to the individual.

Pigmented Naevi

Pigmented naevi, or pigmented moles, develop before or after birth, are of varying size, and are characterized by naevus cells.

Many of the public consider most moles to be dangerous and prone to change into malignant growths. Fortunately, this is rarely true, and what the public alludes to we term a melanoma, for occasionally moles do develop into melanomas which are a particularly malignant form of carcinoma. One of the first considerations in the examination of the mole is to decide whether it is likely to become a melanoma or not.

Before describing the clinical varieties of moles it is valuable to understand the pathological changes that occur to produce benign and malignant moles.

There are, principally, two cells concerned:

1. The melanocytes.
2. The naevus cells.

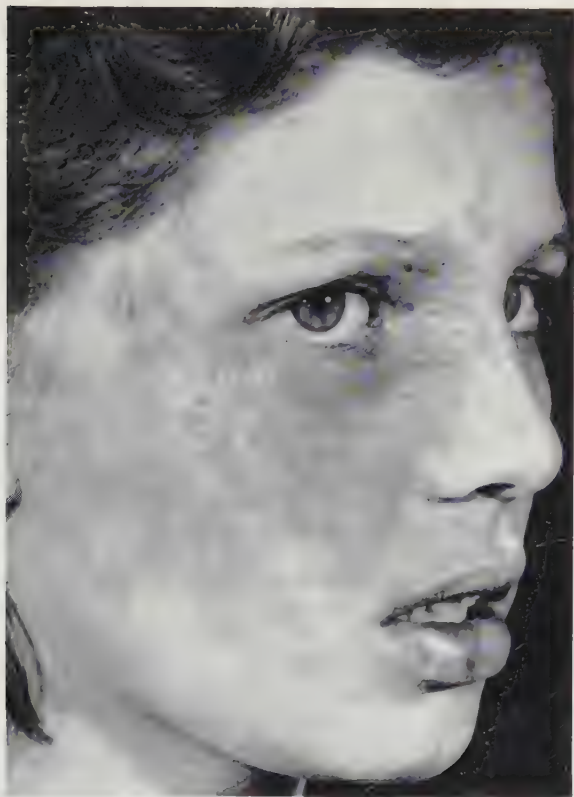


FIG. 69. Naevus flammeus (Institute of Dermatology, University of London).

The *melanocytes* are also known as dendritic or clear cells and are now considered to be neural cells, which originate in the neural crest and migrate to the epidermis during early foetal life. In sections stained with haematoxylin and eosin they are seen as clear cells with a dark-staining nucleus and clear cytoplasm. They lie in the basal

layer. When stained with silver they appear as dendritic cells with long branching processes.

The *naevus cells* are also considered to be of neural origin. The naevus cell is oval or cuboidal in shape, with a pale round or oval nucleus. The cytoplasm is very distinct and the cells contain varying amounts of melanin. But naevus cells vary in appearance, sometimes resembling epithelial cells, at other times histiocytes or other cells.

In sections of naevi, the naevus cells are seen to lie singly, or in nest-like clusters.

From the histological point of view, there are three types of naevus. Decisions on treatment and prognosis can be made from this knowledge.

The three types of naevus are:

1. Junction naevus.
2. Intradermal naevus.
3. Compound naevus.

The *junction naevus* shows active formation of naevus cells at the epidermo-dermal junction only. Naevus cells are also to be seen in the dermis in nest formation, without an inflammatory infiltrate, or scattered singly in the dermis, *with* an inflammatory infiltrate.

In the first case the danger of malignancy is slight; in the second case the danger is considerable.

The *intradermal naevus* shows naevus cells in the dermis without epidermo-dermal junction activity.

The *compound naevus* shows naevus cells in both areas.

CLINICAL FEATURES

The *junction type* of naevus appear at birth or any time afterwards. It is a macule or papule, brown or smooth, and hairless. It may occur anywhere and when on the palms, soles or genitalia is more than likely to be premalignant. For lesions in these areas and any other area, such a supposition can only be substantiated by microscopical examination.

The *intradermal type* is a papule, usually hairy, well defined, smooth, flesh-coloured or brown. It is most common on the face and neck.

TREATMENT

Excision and routine biopsy is the best treatment for pigmented naevi.

Melanoma*Melano-carcinoma*

This rare disease arises from a junction-type naevus (see above), or from normal skin. For clinical features, see Chapter 19.

Warty Naevus

These lesions may be single or multiple, and are brown in colour.

Naevus cells may be absent or not, depending on the presence of an associated pigmented naevus.

Clinically they appear as hyperpigmented patches, or warty streaks following the course of a nerve, and are then often called linear naevi.

TREATMENT

Cauterization or diathermy. Sometimes surgical excision is required.

Bibliography

The following books are amongst those which have been consulted, and are recommended for further reading.

INGRAM J.T. & BRAIN R.T. (1955) *Sequeira's Diseases of the Skin*. J. & A. Churchill, London.

ROXBURGH A.C. revised by BORRIE, PETER (1963) *Common Skin Diseases*. H.K. Lewis, London.

PILLSBURY D.M., SHELLEY M.B. & KLIGMAN A.M. (1960) *Dermatology*. W.B. Saunders, Philadelphia.

WILKINSON D.S. (1961) *The Nursing and Management of Skin Diseases*. Faber & Faber, London.

LEVER W.F. (1961) *Histopathology of the Skin*. Pitman Medical, London.

SCHWARTZ L., TULIPAN L. & BIRMINGHAM D.J. (1957) *Occupational Diseases of the Skin*. Kimpton, London.

The British National Formulary 1966.

ST JOHN'S HOSPITAL FOR DISEASES OF THE SKIN (1950). *The Pharmacopoeia*. Chaseton Press, London.

Examination Questions

Samples of questions set in recent examinations at Oxford, Cambridge, London, Dublin and Edinburgh Universities, for the Final M.B.

Write short notes on the aetiology, distribution, and diagnosis of any three of the following conditions: (1) acne vulgaris; (2) pityriasis rosea; (3) alopecia areata; (4) herpes zoster; (5) xanthoma tuberosum.

Indicate briefly the dermatological features of the following systemic diseases: (1) sarcoidosis; (2) disseminated lupus erythematosus; (3) Hodgkin's disease.

Describe briefly the symptoms and signs of (1) pityriasis rosea; (2) psoriasis; (3) lichen planus.

Describe the rashes in (1) chicken-pox; (2) smallpox; (3) measles. Indicate the important points of difference between them.

Describe briefly: (1) dermatitis herpetiformis; (2) lichen planus; (3) pityriasis versicolor. Give brief notes on their treatment.

Give the main clinical features of: (1) scabies; (2) pityriasis rosea; (3) alopecia areata.

Give the symptoms and signs of secondary syphilis.

Describe the cutaneous manifestations of: (1) neurofibromatosis; (2) pellagra; (3) smallpox.

Name five causes of erythema nodosum, and give one other characteristic feature of the disease in each case.

Give two clinical points of distinction between each of the following pairs of skin lesions: (1) lupus vulgaris and lupus erythematosus; (2) smallpox and chicken-pox; (3) measles and rubella; (4) pityriasis rosea and psoriasis; (5) erythema nodosum and urticaria; (6) exfoliative dermatitis and eczema; (6) acne vulgaris and impetigo.

Approximate Metric and Imperial Equivalents

Weight

1 mg = .016 gr

1 g = 15 gr

= 0.03 oz (avoir, apoth)

= 0.25 dr

1 kg = 2.2 lb

1 gr = 60 mg

1 dr = 4 g

1 oz = 30 g

1 lb = 0.45 kg

Fluid Measure

1 ml = 15 minim

= 0.04 fl oz

1 litre = 35 fl oz

= 2 pints

= 0.22 gallons

1 m = 0.06 ml

1 fl oz = 30 ml

1 pint = 0.5 litre

1 gal = 4.5 litre



Index

- abdomen, lesions of 20
acantholysis 10
acanthosis 10
acne conglobata 189
acne keloid 189
acne vulgaris 187-92
 treatment 190-1
acquired alopecia 202
acromegaly 168
acuminate wart 136
Addison's disease 169
adult-life eczema 40
albinism 163
allergic eczema 39
alopecia 201-7
 treatment 206-7
alopecia areata 203-5
alopecia totalis 205
alopecia universalis 205
ano-genital area, lesions of 20
ano-genital pruritus 98-9
anthrax 118
antibiotics 33
 effects on skin 93
antihistamines 34
anti-malarials 34
antipruritics 30
antiseptics 30
ants 161
apocrine glands 3
arms, lesions of 20
arsenic, effects on skin 93
asiatic smallpox 144
athlete's foot 121-3
atopic dermatitis 39
atopic eczema 39-45
 treatment 43-5
atopy 39
atrophy 18
avitaminosis 173
axillae, lesions of 19

back lesions 20
baldness 202-3
barbiturates, effects on skin 93
basal cell carcinoma 220-4
basal cell epithelioma 220-4
basal cells 3
basal layer 3
baths, treatment 26
Bazin's disease 117
bedbug 161
bees 161
Besnier's prurigo 39
biopsy 21-2
blood defects, in purpura 175
blood vessels, dermis 7
boil 106
bran bath 26
bromides, effect on skin 93
bullae 17
bullous diseases 148 *et seq.*
bullous eruptions, differential diagnosis of 154

calamine lotion 27
cancer, skin changes related to 230
candidiasis 129-30
capillary wall defects in purpura 173-5
carbon dioxide treatment 35
carbromal, effect on skin 93
carbuncle 106

- carcinoma of nipple 226
- carotene 9
- cautery 35
- cavernous haemangioma 235-6
- chest lesions 19
- childhood-type eczema 40
- cheilopompholyx 46
- chloroquine, effect on skin 93
- chlorpromazine, effect on skin 93
- chilblains 95
- chicken-pox 143-4
- chloasma 162
- cheilitis 195
- clothing, as cause of dermatitis 48-51
- collagen fibres 6
- compound naevus 238
- common wart 134, 136
- congenital alopecia 202
- congenital and hereditary diseases 231-9
- connective tissue fibres 6
- contact dermatitis 47-57
- Coomb's test 180
- cosmetics and dermatitis 51
- cowpox 147
- creams 29-30
- crusts 17
- Cushing's syndrome 168
- cysts, sebaceous 192
- dermis 6-9
 - blood vessels 7
 - cellular elements 7
 - connective tissue fibres 6
 - lymphatic vessels 8
 - muscles of 8
 - nerves of 8
 - water in 8
- depigmentation 18
- dermabrasion 37
- dermatitis, contact 47-57
 - cause 47-8
 - characteristics of substances causing 48-54
 - exfoliative 76-8
 - seborrhoeic 71-3
 - treatment 56-7
 - varicose 170-3
- dermatitis, differential diagnosis of
 - common scaly conditions 78
- dermatitis artefacta 101-2
- dermatitis herpetiformis 81, 148-50
 - treatment 149
- dermatitis venenata 47
- dermatology
 - terminology 10
 - treatment 23-38
- dermatomyositis 186
- detergents and dermatitis 51
- dhobie itch 126-7
- diabetes mellitus 168
- diagnosis, general approach to 11-12
- diathermy 35
- discoïd eczema 45
- disease, cellular elements in 7
- diseases, general classification 22
- diseases of unknown cause, systemic 176-86
- disseminated neurodermatitis 39
- dressings 25-6
- drug eruptions 91-3
- drug purpura 175
- drugs as cause of purpura 173, 174
- dysidrosis 46-7
- ears, lesions of 19
- eccrine glands 3
- eczema, atopic 39-45
- eczema herpeticum 141
- eczema vaccinatum 42, 147
- elastic fibres 6
- emulsifying bath 26
- endocrines 34
- eosinophils 7
- epidermis 1-6
 - appendages of 3-6
- epithelioid cells 7
- erythema, toxic 79
- erythema induratum 117
- erythema multiforme 79-83
 - treatment 82
- erythema nodosum 83
- excoriated acne 188

- exfoliative dermatitis 76-8
- exfoliative pemphigus 151
- extra-mammary Paget's disease 226
- eyelids, lesions of 19
- face lesions 19
- feet lesions 20
- fibres, connective tissue 6
- fibroblasts 6, 7
- fibroma 213
- filiform wart 135, 136
- fingers, clubbing of 200
- fixed cells 7
- fleas 160-1
- foam cells 7
- folliculitis barbae 107-8
- freckles 162
- fungal diseases 119 *et seq.*
- furuncle 106
- german measles 147
- giant urticaria 86
- glossitis, superficial 130
- granular layer 3
- granuloma pyogenicum 217-18
- granuloma telangiectaticum 217-18
- Grenz rays 37
- griseofulvin 34
- guarnieri bodies 131
- haemangiomas 234-6
- haemoglobin, reduced 3
- hair 4-5
 - cyclical growth of 5
 - diseases of 201-9
 - function of 5
 - greying of 209
 - ingrowing 209
- hair growth 4
 - anomalies of 209
- hands, lesions of 20
- health, cellular elements in 7
- Henoch-Schoenlein syndrome 173, 174
- herpes simplex 140-1
- herpes zoster 137-40
- Hippocratic fingers 200
- hirsuties 207-8
- histiocytes 7
- histology of skin 1-9
- history-taking 12 *et seq.*
- Hodgkin's disease 229
- hormonal disorders 168-9
- hormones and acne 187-8
- horny layer 3
- hyperidrosis 193-4
- hyperkeratosis 10, 18
- hyperpigmentation 18, 162
- hyperthyroidism 168
- hypertrophy 10
- ichthyosis 231-4
- idiopathic thrombocytopenic purpura 175
- impetigo 104-6
- infantile acne 188
- infantile eczema 40
- intertrigo 130
- intra-dermal naevus 238
- iodides, effect on skin 93
- junction naevus 238
- Kaposi's varicelliform eruption 141
- keloids 211-13
- keratin 3, 8
- kerato-acanthoma 218-19
- keratosis
 - of palms and soles 234
 - senile 215-17
- Klebs-Loeffler bacillus 104
- Koebner's phenomenon 65, 75
- koilonychia 200
- Koplik's spots 146
- Krause's end bulbs 8
- Kveim test 183
- laboratory tests 21
- Langhans giant cells 7

- lanugo hairs 4
- legs, lesions of 20
- lesions
 - caused by offending substances, distribution of 55
 - identification of 14-21
 - regional distribution of common 19-20
 - summary of types 16-18
- leucocytes 7
- leucoderma 163
- leuconychia 200
- leucoplakia 196-7, 219
- leukaemia 229-30
- Libman-Sacks syndrome 181
- lichen planus 73-6
 - treatment 73
- lichen planus bullosus 75
- lichen planus hypertrophicus 75
- lichen simplex chronicus 99-100
- lip lesions 19, 195
- lipids, skin surface 8
- lipoma 215
- lipschütz bodies 131
- lotio alba 27
- lotio hydrag. 28
- lotions 27-8
- lues 108-14
- lupus erythematosus 10, 176-82
 - acute 180-2
 - chronic 176-80
- lupus vulgaris 115-17
- lymphatic vessels of dermis 8
- lymphocytes 7

- macrophages 7
- macules 16
- malpighian layer 3
- measles 146
 - german 147
- medicines, effects of, on dermatitis 53
- Meissner's tactile bodies 8
- melanin 8, 9
- melanocytes 3, 237-8
- melanoid 9
- melanoma 239
 - malignant 227-9
- Merkel-Ranvier discs 8
- metabolic disorders 166-7
- metastatic carcinoma 230
- migrating cells 7
- miliaria 95
- moles
 - cells of 236
 - pigmented 236-9
- molluscum contagiosum 142-3
- molluscum sebaceum 218-19
- monilethrix 209
- moniliasis 129-30
- mouth lesions 19, 196-8
- mucous membranes 194-8
- muscle, dermis 8
- mycosis fungoides 227
- myxoedema 168

- naevi, pigmented 163, 236-9
- naevus
 - types of 238
 - warty 239
- naevus cells 238
- naevus flammeus 236
- nails 5-6
 - affections of 198-201
 - brittle 200
 - hypertrophy of 200
 - ingrowing 199-200
 - pigmentation of 201
 - shedding of 201
 - spoon-shaped 200
- napkin dermatitis 50, 51
- neurodermatitis, localized 99-100
- neurofibroma 213-15
- neurotic excoriations 103
- nerves, dermis 8
- nipple, Paget's disease of 225-6
- nodules 17
- nummular eczema 45

- occupational acne 190
- occupational causes of dermatitis 54
- oidiomycosis 129

- ointments and pastes 28-9
- onychia 129
- onychogryphosis 200
- oral lesions 195-7
- orf 147
- oxyhaemoglobin 8
- Pacinian corpuscles 8
- Paget's disease of nipple 226
- paints 31
- papules 16
- papulo-necrotic tuberculid 117
- parakeratosis 10
- parasites, diseases due to 155 *et seq.*
- paronychia 129, 198-9
- pastes, ointments and 28-9
- patch test 21
 - for contact dermatitis 55
- pediculosis 158-9
- pediculosis capitis 159-60
- pediculosis corporis 160
- pediculosis pubis 160
- pedopompholyx 46
- pellagra 167
- pemphigoid 152
- pemphigus 150-2
 - treatment 152
- pemphigus erythematodes 152
- penicillin, effect on skin 93
- perlèche 130, 195
- pernio 95
- pigmentation, disturbances of 162-5
- pili incarnati 209
- pityriasis rosea 66-71
 - clinical features 68
 - treatment 69
- pityriasis versicolor 129
- plane wart 134, 136
- plantar wart 134
- plants and dermatitis 53
- plaques 18
- plasma cells 7
- Plummer-Vinson syndrome 200
- polymorphonuclear leucocytes 7
- polythene coverings 26
- pompholyx 46-7
- potassium permanganate bath 26
- powders 26-7
- pregnancy, skin lesions and 169
- prickly heat 95
- primary lesions 14-15
- pruritus 98
- psoriasis 58-66
 - causes 58-60
 - clinical features 61-5
 - pathology 60-1
 - prognosis 66
 - treatment 65-6
- psychotherapy in atopic eczema 44
- purpura 173-5
- pustules 17
- Raynaud's disease 186
- Raynaud's phenomenon 185
- reticulum fibres 6
- ringworm
 - of foot 121-3
 - of groin 126-7
 - of hands 123-4, 198
 - of nails 124-5
 - of scalp 127-8
 - of the body 125-6
- rodent ulcer 220-4
- rosacea 88-90
- rubella 147
- sarcoidosis 182-4
 - treatment 184
- scabies 155
 - treatment 157
- scalp lesions 19
- scars 17
- scleroderma 10, 184-6
 - localized 184-5
 - systemic 185-6
 - treatment 186
- scurvy 173
- sebaceous cysts 192
- sebaceous glands 4
- seborrhoea 192
- seborrhoeic dermatitis 71-3
- seborrhoeic warts 210-11
- secondary lesions 15-16
- sedatives 35

- senile pruritus 98
- senile purpura 173, 174
- serum, effect on skin 93
- shingles 137-40
- simple haemangioma 234-5
- simple purpura 173, 174
- skin
 - absorption by 9
 - histology 1-9
 - pathology of 9-10
 - pigmentation of 8
 - qualities of 1
 - sensation types 9
- skin diseases
 - bacterial 104 *et seq.*
 - nail changes in 201
 - resulting from emotional upsets 97
 - resulting from psychological disorders 97
- smallpox 144-6
- sodium bicarbonate in bath 26
- spongiosis 10
- squamous cell carcinoma 224-5
- steroid lotions 27
- steroids 32-3
 - effect on skin 93
- Stevens-Johnson syndrome 81, 82
- stomatitis aphthous 196
- Sucquet-Hoyer canal 7
- sunburn 94
- sweat glands 3, 193-4
- syphilis 108-14
 - clinical features 109, 110
 - congenital 109, 114-15
 - lesions 111
 - treatment 114
- syringomyelia 186
- systemic diseases 175
 - and purpura 173, 174
- systemic therapy for atopic eczema 44
- tar bath 26
- thorium x 37
- thrombophlebitis, treatment for 172
- thrush 129-30
 - intra-oral 130
- tinea 120-1
 - tinea capitis 127-8
 - tinea circinata 125-6
 - tinea corporis 125-6
 - tinea cruris 126-7
 - tinea manuum 123-4
 - tinea pedis 121-3
 - tinea unguium 124-5
 - tinea versicolor 129
- toxic erythema 79
- traction alopecia 206
- treatment
 - acne vulgaris 190-1
 - alopecia 206-7
 - atopic eczema 43-5
 - contact dermatitis 56-7
 - dermatitis herpetiformis 149
 - erythema multiforme 82
 - exfoliate dermatitis 77-8
 - external 25-31
 - hirsuties 208
 - internal 32-5
 - lichen planus 73
 - lupus erythematosus, acute 182
 - lupus erythematosus, chronic 180
 - nummular eczema 45
 - pemphigus 152
 - physical agents 35-8
 - pityriasis rosea 69
 - pompholyx 46-7
 - psoriasis 65-6
 - sarcoidosis 184
 - scabies 157
 - seborrhoeic dermatitis 71-2
 - syphilis 114
 - tinea 121
 - tinea pedis 123
 - ulcers 172-3
 - varicose dermatitis 172-3
- trichotillomania 102-3
- tuberculosis 115
- tuberculosis verrucosa 118
- tumours
 - benign 210-19
 - malignant 220-30
- tylosis 234
- ulcer, varicose dermatitis and 170-3

- ulcers 17
 - treatment 172-3
- ultra-violet rays 36
- urticaria 81, 84-8
 - bullous 86
- vaccinia 147
- vaginitis 130
- varicella 143
- varicose dermatitis 170-3
 - treatment 172-3
- variola 144-6
- vascular disorders, diseases due to 170 *et seq.*
- vegetating pemphigus 151
- vegetative lesions 18
- venous-stasis purpura 173, 174
- verruca plana 134
- verruca vulgaris 134
- vesicles 17
- viruses, diseases due to 131 *et seq.*
- vitamin deficiencies 167
- vitamins 34
- vitiligo 163
- warts 131-6
- warty naevus 239
- wasps 161
- water in skin 8
- wheals 16
- xanthoma planum 166
- xanthoma tuberosum 166
- xanthomata 166
- X-rays 36-7

Textbook of Dermatology

Edited by ARTHUR ROOK M.A. M.D. F.R.C.P.
F.Z.S., D.S. WILKINSON M.D. F.R.C.P. and F.J.
EBLING M.SC. PH.D. June 1967. 1,200 pages,
512 illustrations. About £10

A Course in Renal Diseases

By G.M. BERLYNE M.B. M.R.C.P. 1966. 412
pages, 71 illustrations. 37s 6d

Essentials of Fluid Balance

By D.A.K. BLACK M.D. F.R.C.P. *Third Edition*,
1964. 180 pages, 7 illustrations. 25s

The Autonomic Nervous System

By J.H. BURN M.D. F.R.S. *Second Edition*, 1965.
144 pages, 7 illustrations. 18s 6d

Clinical Haematology in Medical Practice

By G.C. DE GRUCHY M.D. F.R.A.C.P. M.R.C.P.
M.C.P.A. *Second Edition*, 1964 (*Reprinted*
1966). 696 pages, 79 illustrations. 70s

A Guide to Psychiatry for Students of Medicine

By JOHN GIBSON M.D. D.P.M. 1963. 256 pages.
27s 6d

Immunology for Students of Medicine

By J.H. HUMPHREY M.D. B.CHIR. F.R.S. and
R.G. WHITE M.A. D.M. B.CH. *Second Edition*,
1964 (*Reprinted* 1965, 1966). 512 pages, 91
illustrations. 47s 6d

Diseases of Children

By HUGH JOLLY M.A. M.D. M.R.C.P. D.C.H. 1964
(*Reprinted* 1966). 596 pages, 110 illustrations
(2 colour). 47s 6d

An Introduction to Electrocardiography

By L. SCHAMROTH M.B. B.CH. M.R.C.P.E.
M.R.C.P. (Glas.). *Third Edition*, 1966. 208
pages, 191 illustrations. 20s

A Guide to Orthopaedics

By T.T. STAMM M.B. B.S. F.R.C.S. *Second*
Edition, 1964. 128 pages, 20 illustrations.
18s 6d

Diseases of the Digestive System

By S.C. TRUELOVE M.A. M.D. F.R.C.P. and P.C.
REYNELL M.A. D.M. F.R.C.P. 1963 (*Reprinted*
1965). 704 pages, 205 illustrations. 84s

THE 'LECTURE NOTES' SERIES

This series, now well known throughout the world, was not originally planned to embrace many volumes, but rather allowed to grow by demand. The books in the series have not been published in the belief that medicine can be adequately presented in a formal series: they are simply intended to set out the essentials of a subject concisely and lucidly, and to provide for the student both an introductory guide and an aid to rapid revision before examinations. For the general practitioner also they afford a convenient source of guidance on any problem he may meet in his daily work. The following titles in the series are already published or in production.

Gynaecology by Josephine Barnes M.A. D.M. M.R.C.P. F.R.C.S. F.R.C.O.G. 1966. 248 pages, 30 illustrations. 22s 6d

Neurology by Ivan T. Draper M.B. CH.B. M.R.C.P. 1965. 240 pages, 29 illustrations. 18s 6d

General Surgery by Harold Ellis D.M. M.CH. F.R.C.S. and Roy Calne M.S. F.R.C.S. 1965. 392 pages, 35 illustrations. 42s

Psychiatry by James Willis M.B. M.R.C.P. D.P.M. 1964 (*Reprinted* 1966). 100 pages. Paper, 7s 6d

Pathology by A.D. Thomson M.A. M.D. M.R.C.P. and R.E. Cotton M.D. B.S. 1962 (*Reprinted* 1963, 1965). 1,056 pages, 39 illustrations. 55s

Ophthalmology by P.D. Trevor-Roper M.A. M.D. B.CHIR. F.R.C.S. D.O.M.S. *Second Edition*, 1965. 116 pages, 78 illustrations (12 colour). 16s

Obstetrics by Frank Musgrove M.D. F.R.C.O.G. D.A. *Second Edition*, 1966. 432 pages, 24 illustrations. 25s

Pharmacology by J.H. Burn M.D. F.R.S. *Eighth Edition*, 1965. 160 pages. 12s 6d

Diseases of the Ear, Nose and Throat by E.H. Miles Foxen F.R.C.S. D.L.O. 1961 (*Reprinted* 1964, 1965). 196 pages, 96 illustrations. 18s 6d

The Use of the Microscope by R. Barer M.C. M.A. M.B. B.SC. B.S. *Second Edition*, 1956 (*Reprinted* 1959, 1963). 84 pages, 8 illustrations. Paper, 7s 6d

BLACKWELL SCIENTIFIC PUBLICATIONS · OXFORD